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RISK ASSESSMENT OF WASTEWATER DISINFECTION

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FOREWORD

The U.S. Environmental Protection Agency is charged by Congress with protecting the Nation's land, air, and water systems. Under a mandate of national environmental laws, the agency strives to formulate and implement actions leading to a compatible balance between human activities and the ability of natural systems to support and nurture life. The Clean Water Act, the Safe Drinking Water Act, and the Toxics Substances Control Act are three of the major congressional laws that provide the framework for restoring and maintaining the integrity of our Nation's water, for preserving and enhancing the water we drink, and for protecting the environment from toxic substances. These laws direct the EPA to perform research to define our environmental problems, measure the impacts, and search for solutions.

The Water Engineering Research Laboratory is that component of EPA's Research and Development program concerned with preventing, treating, and managing municipal and industrial wastewater discharges; establishing practices to control and remove contaminants from drinking water and to prevent its deterioration during storage and distribution; and assessing the nature and controllability of releases of toxic substances to the air, water, and land from manufacturing processes and subsequent product uses. This publication is one of the products of that research and provides a vital communication link between the researcher and the user community.

Cost-effectiveness and environmental assessment are the two criteria most commonly used to select among alternative problem solutions; however, when substantive risk is created by alternative solutions, risk assessment becomes a third decision criterion. The research presented in this publication illustrates the application of risk assessment methods to wastewater disinfection, a problem where all alternatives create human and ecological risks.

Francis T. Mayo, Director
Water Engineering Research Laboratory

ABSTRACT

A risk assessment data base is presented for several wastewater disinfection alternatives, including chlorination, ozonation, chlorination/dechlorination, and ultraviolet radiation. The data base covers hazards and consequences related to onsite use and transportation of the disinfectants and ultimate disposal of disinfected effluents. A major segment of the data base deals with the effects of chlorination products in aquatic ecosystems. Energy consumption and cost analyses are also presented for chlorination and ozonation. Example risk calculations are presented for two hypothetical wastewater treatment plants. The usefulness of the data base for risk assessment is also discussed.

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SECTION 1

INTRODUCTION

Risks in today's world have been the center of growing attention. Increased risk awareness in modern technological societies is an outgrowth of technologic development and economic achievement. Increased risk awareness may also be the result of risk overload, a condition in which the maximum tolerance for risk is being taxed.

Regardless of the reasons for an increased risk awareness, technologies with inherent risks (air, water and soil pollution, for example) cannot continue to develop without considering the net impact of those inherent risks on humans, other living organisms, and the environment. Also, cost-effective public policy cannot be formulated without adequate assessment of risk. Finally, because the world will never be free of risk, public policy should be formulated with some consideration for its management.

Effective risk management is based on a qualitative and quantitative understanding of the risks associated with public policy decisions. Risk assessment provides that understanding and is the first step in risk management.

In 1979, faculty members at the University of Colorado at Denver undertook the development of a wastewater disinfection risk assessment because a controversy raged over whether wastewater effluents should be disinfected and, if so, which method was preferred. Since all of the alternatives included inherent risks, the wastewater disinfection question was an appropriate application of risk management.

A risk assessment of all alternatives seemed unnecessary and inappropriate because so many disinfectants are unlikely to be used. Thus, the assessment focused on chlorination and those disinfection processes that appeared most likely to replace chlorination. The alternatives selected for the risk assessment were as follows:

1. Chlorination,
2. Chlorination followed by dechlorination,
3. Ozonation,
4. Ultraviolet radiation, and
5. No disinfection

The choice of a disinfectant should be made at a regional or local level because key variables such as the length of the chlorine haul are site specific. Furthermore, since the water quality management structure evolving in the United States contains a strong emphasis on local and regional planning and policy making, the use of this risk assessment data base in evaluating disinfection alternatives is likely to occur at those government levels. Thus the specific products expected from this risk assessment are designed for local public policy-setting applications. The two primary products are:

1. The collection and evaluation of a data base, and
2. The development of a method for using that data base in a wastewater disinfection risk assessment.

A preliminary analysis of the risks associated with wastewater disinfection revealed the broad mix of expertise required to investigate such risks. Thus the study team was designed to be interdisciplinary. Most of the work reported here was developed by a physicist, a chemist, a biologist, an economist, and a civil engineer.

SECTION 2

CONCLUSION

A risk analysis is a detailed examination performed to understand the nature of unwanted, negative consequences to human life, health, property, or the environment. The purpose of this analytical process is to provide information regarding the nature and frequency of these negative consequences. The methods used depend on the objectives, the data available, and the resources. The analysis can range from a back-of-the-envelope calculation to a fault-tree analysis of each element in the system(s). Each risk calculation has an associated confidence interval that may or may not be explicit in the calculations.

In most if not all such analyses, resource constraints and data gaps make quantitative estimates of some risks impossible. Such was the case with the study reported here. Nevertheless, some useful conclusions have emerged.

1. Though wastewater treatment plants have a poor overall safety record, with an accident rate similar to that of metal mining, exposure to toxic substances (primarily chlorine) accounts for only 4% of these accidents. Note, however, that data on the relative severity (e.g. lost workdays) of these exposures are not available. Thus though the rate is similar to that for insect bites, the risk (rate x severity) may not be similar. In addition, the possibility of a low-probability, high-consequence event (i.e., massive exposure) cannot be discounted but is impossible to quantify without a more detailed analysis (i.e., a fault tree).

2. Most chlorine is shipped by rail (85%). The bulk of the rest is shipped by tank truck (9.9%) or by common carrier as cylinders (114 kg or 0.9 metric ton). Though railroads generally have a much better safety record than truck shipments (particularly in 114-kg cylinders), the Youngstown, Florida, accident in February 1978 illustrates the possiblity of low-frequency, high- consequence events for railroad

shipment. In this case, there were 8 fatalities, 260 injuries, more than $1 million in property damage, and a release of 45,400 kg of chlorine gas.

3. A review of the literature on aquatic toxicology of chlorine indicates that for all byproducts (e.g. chloroform) except total residual chlorine (TRC) the observed effluent levels are below those known to be acutely toxic. Thus only TRC will lead to an acute response, and then only if the dilution in the receiving stream is insufficient to lower the resulting level sufficiently. These effects can range from avoidance to death of aquatic organisms. Further studies are needed to determine whether TRC and various byproducts could lead to chronic effects in aquatic organisms at the levels encountered.

4. The compounds found in effluents are well below the acute toxicity levels for humans. In general, the contribution to finished drinking water from wastewater disinfection will be much smaller than from drinking water treatment. Though chloroform and trichloroethylene are carcinogens and (assuming no threshold) will therefore contribute some additional risk of cancer to humans, this contribution is relatively small (less than one excess case of cancer for every 50,000 persons exposed to 5 ug/L of chloroform over a lifetime). This assumes no dilution of the effluent or subsequent loss. Since drinking water chlorination is the major cause of human exposure to byproducts of chlorination, these risks are not expected to be an important consideration for wastewater disinfection.

5. Though it is possible to identify hazards associated with the alternatives of chlorination-dechlorination, ozonation, and UV disinfection, the lack of data made quantitation impossible. In general, these methods are less of a concern for the environment and the general public than chlorination. However, accidental releases of SO_2 (used in dechlorination) can pose hazards to humans and terrestrial and aquatic organisms. Ozone poses a risk to workers in the plant and to vegetation in the vicinity of the facility. Though Europe has had considerable experience with ozone disinfection of drinking water, no data on human risks are readily available. Although ozone is toxic to aquatic life, its lack of stability in water makes that risk minimal. Even less information is available on the risk of ultraviolet disinfection. The primary hazards are from human exposure to the radiation itself (burns),

exposure to ozone produced by the radiation, and exposure to high electrical voltages (a hazard also present with ozonation).

6. The risks of not disinfecting wastewater were also analyzed. There was no discussion in the scientific literature on the effect on aquatic organisms, though it might be assumed that increased nutrients could have some detrimental effects. The human hazards, of course, relate to exposure to pathogenic organisms. The most common risk would be gastrointestinal illness, although there is also some risk of exposure to life-threatening or disabling organisms as well.

7. An analysis was also performed on the energy use and cost of chlorination versus ozonation. The capital costs are about twice as great for ozone as for chlorine, and operating and maintenance costs are, in the best case, 35% higher for ozone than chlorine. However, wide variation exists in the operation and maintenance costs for ozone, depending on the efficiency of ozone generation and absorption and the cost of energy. Note, however, that the cost of disinfection is only a few per cent of the total cost of wastewater treatment, resulting in a maximum difference of 10% for the total cost. The energy use for chlorine was found to be one tenth that for ozone (and similar to that for UV disinfection), but only on-site energy use was taken into account. Though the original generation of chlorine is, in fact, energy-intensive, the fact that chlorine use for disinfection is such a small portion of the total chlorine use is likely to lead to a disproportionately smaller share of increased energy costs.

8. A risk assessment data base for the wastewater disinfection alternatives of chlorination, ozonation, ultraviolet radiation, chlorination/dechlorination, and no disinfection has been collected and reported here. Portions of the chlorination data base are tabulated in the Appendices, and the sources of the data base are listed in the References section of this report. The data base is heavily skewed towards the chlorination alternative. This imbalance creates an excessive amount of attention on the hazards of chlorination and may create the illusion that the other alternatives involve less risk. In addition, the nature of the data base is not well suited to quantitative risk assessment because many of the data do not support the development of dose-response relationships for many acute responses and for essentially all chronic responses.

9. The study's goal of developing a risk assessment tool for use by local and regional policy makers in evaluating the desirability of requiring wastewater disinfection has not been realized because the available data base is insufficient to support such analyses. Fault tree analyses could possibly fulfill that goal, but that project must be undertaken in future research projects.

SECTION 3

RISK ASSESSMENT METHODS

Risk assessments consist of several parts, but they do not all contain the same parts. The elements in each risk assessment are governed by the nature of the risk being assessed, the quantity and quality of available data, and the level of risk assessment effort that can be supported by the available resources.

The several parts of a risk assessment are designed to satisfy one or more of the following objectives:

1. Completely define the potential hazards associated with the subject activity.
2. Define and quantify the relationship between exposure and severity for each of the hazards defined in item 1 above.
3. Estimate the frequency and/or probability of exposure for each of the identified hazards.
4. Synthesize the results of the above three tasks into a risk model.

While the first objective realized must be item 1 above, the other three objectives are not necessarily pursued or realized in the order shown. For example, the type of data available is usually discovered while pursuing items 2 and 3, and the results of those tasks will determine the type and complexity of the risk model chosen in item 4. However, the type of model chosen will provide the focus for the data searches in items 2 and 3. This feedback relationship requires a risk assessment to use an iterative method in order to achieve the highest level of clarity.

The hazard definition tasks are qualitative,and are designed to answer such questions as, "What happens if humans (or fish...or plants..etc.) are exposed to gaseous chlorine (or dissolved chlorine...or chlorine reaction products...or ozone...etc.)?". These questions can usually be answered through laboratory or field studies and/or through extensive literature searches. There have been many studies designed to answer this type of question so the literature search method can be very fruitful. Sometimes it is necessary to identify intermediate causes of hazards before this task can be completed. In this assessment, hazards caused by process reaction products and the identification of those hazards could not be accomplished until the reaction products were identified. Therefore, some of the hazard identification sections in this report also contain a discussion of the expected process reaction products.

The quantitative elements of a risk assessment are produced by the severity and frequency definition tasks. When dealing with hazards caused by chemical agents, the severity definition element usually involves the discovery or development of dose-response models. Since chlorine, ozone, and the process reaction products all fall into the "chemical agent" category, a major part of this study involved the search for dose-response models.

The frequency definition task is dependent on the nature of the available data. For example, transportation accident data are often reported in terms of ton-miles, so the frequency definition task must estimate the expected increase or decrease in ton-miles resulting from a change in policy or practice. This estimate can then be transformed into expected accidents. Sometimes the frequency definition task cannot be accomplished in a global or generalized context. In those cases the frequency definition task has been omitted from this study due to the narrowness of the study scope.

The frequency definition task may also depend on the chosen risk model. For example, if a risk model is chosen where the hazard (or outcome) is a function of event A then the frequency definition task must find means of predicting the occurrence of event A. On the other hand, if the hazard is modeled as a function of some measureable but not necessarily causative quantity, such as man-years of labor, then the frequency definition task is the same as in the paragraph above. The dependence of the frequency definition task on the choice of risk model should be clarified in the following discussion of risk models.

Risk assessments have developed many ways of modeling risk; however, all of the risk models attempt to do one thing: predict the probability of realizing a hazard (or outcome) Y as a function of an event A (or events A, B, C, etc....). Ideally the model should be based only on cause and effect relationships, i.e. hazard Y should be caused by event A. This model might be shown mathematically as:

$$Y = f(A)$$

Such a simple relationship seldom occurs in natural systems.

Usually the event A is also a function of an earlier event A_1, and that event in turn is a function of an even earlier event A_2, and so forth. This latter model would take the form:

$$\begin{aligned} Y &= f(A), \\ &\text{Where:} \\ A &= f(A_1), \\ A_1 &= f(A_2), \\ \cdot\cdot &\quad \cdot\cdot \\ \cdot\cdot &\quad \cdot\cdot \\ A_{n-1} &= F(A_n). \end{aligned}$$

The conversion of this latter cause and effect model to a probability model leads to:

$$\langle Y\rangle = \langle\langle A/A_1\rangle\langle A_1/A_2\rangle\ldots\ldots\langle A_{n-1}/A_n\rangle\rangle\langle A_n\rangle$$

where $\langle Y\rangle$ is the probability that hazard Y will occur, and $\langle A/A_1\rangle$, $\langle A_1/A_2\rangle$, ..., $\langle A_{n-1}/A_n\rangle$ are the probabilites that each of the causative events will occur given that the previous event has occurred, and $\langle A_n\rangle$ is the probability that the initial event occurred.

This sequential event probability model is frequently used in risk assessment because it usually is easier to define or estimate the relationship between each pair of sequential events than to define the relationship between the initial event $\langle A_n\rangle$ and the resultant hazard $\langle Y\rangle$. This type of model requires that all of the intermediate cause and effect relationships and the severity and frequency elements of those relationships be defined during the risk assessment.

The type of model becomes even more complex and costly if hazard Y can be caused by more than one string of sequential events. This more complex probability model for two strings of sequential events would be:

$$\langle Y\rangle = \langle A/A_1\rangle\ldots\ldots\langle A_n\rangle + \langle B/B_1\rangle\ldots\ldots\langle B_n\rangle,$$

where the term definitions are analagous to the equation above. Further complexity is added when some of the sequential events are also caused by more than one string of earlier events.Some systems also contain synergistic and antagonistic relationships between parallel event strings. Feedback relationships may also exist.

These sequential event cause and effect models can be plotted graphically. The direction of the effect and feedback relationships are shown by lines and arrows, and the synergism and antagonism elements are shown with Boolean logic symbols. The resulting plot resembles a tree, and this visual effect led to the identification of this model as "fault tree analysis".

The high cost of fault tree analysis is usually justified when existing data are not sufficient to construct a probability relationship between the initial event(s) and the hazard Y. Fault tree analysis permits the construction of such a probability relationship by estimating probabilities for several simpler and probably better known relationships. Analogies are often used to estimate these simpler probability relationships.

However, because the funds available were not sufficient to produce a fault tree analysis, this risk assessment was limited to the simplest risk model described above. The funding constraint also eliminated consideration of risks associated with production of the disinfectants at sites other than the wastewater treatment plant site and consideration of indirect risks such as the risks associated with the production of power to operate the disinfection processes. These constraints resulted in a different product than had first been envisioned.

SECTION 4

CHLORINE

HAZARD IDENTIFICATION

On-site Use Hazards

Although the use of chlorine to disinfect wastewater effluents is an effective mechanism for the destruction of pathogens to accepted levels, various risks or hazards can be attributed to chlorine. In addition, there is a growing concern and awareness about the presence of chlorinated chemicals and their associated risks in wastewater. Chlorine is of obvious concern because of its widespread use as a wastewater disinfectant. The identification of risks associated with chlorine as a wastewater disinfectant can be grouped according to production, transportation and handling, and use. Secondary risks associated with power consumption in the production phase and fuel consumption in the transportation phase can also be identified. The use of chlorine as a wastewater disinfectant has very little effect on the production level of chlorine, so if chlorine were totally eliminated as a wastewater disinfectant, the decrease in risk associated with the overall production of chlorine would be small. Secondary risks by their very nature have less significance. So neglecting these production and secondary risks will not have a major impact on the overall risk analysis project.

The primary risks associated with the handling of chlorine are:

1. Human exposure to liquid chlorine
2. Human exposure, both occupational and public, to gaseous chlorine
3. Vegetation exposure to either liquid or gaseous chlorine

Exposure to liquid chlorine is possible for the occupational workforce and can result in severe skin or eye burns. However, the most common exposure is to gaseous chlorine, which is the normal chlorine state at atmospheric pressure and normal temperatures.

The most important human exposure routes to gaseous chlorine are inhalation followed by eye and skin contact. The odor threshold of chlorine is approximately 0.2 ppm (parts-per-million by volume). Below 1 ppm there is little dose-response correlation for workers chronically exposed to chlorine, while chronic exposure to 5 ppm chlorine can bring about respiratory complaints, nausea, increased susceptibility to tuberculosis, and corrosion of the teeth (NAS, 1976). Acute exposure to chlorine presents immediate and latent effects. Immediate effects begin with throat and mucuous membrane irritation for an exposure of one hour at 7 ppm. Higher concentrations lead to cough, conjunctivitis, pulmonary edema, and death. A 100 ppm exposure is lethal in only a few seconds, representing a significant risk to humans for an accidental release of gaseous chlorine. Latent effects are less pronounced and often difficult to diagnose but may include broncospasm, especially in asthmatic people, and difficult or painful breathing (NAS, 1976).

There is a wide range of sensitivities among vegetation species when exposed to gaseous chlorine. Chlorine exposures result in spotting of vegetation at low concentrations to decay and death of the plant at higher concentrations. Threshold concentrations for acute injury vary but typically begin in the 0.5 to 1 ppm range for one hour exposures. The pattern of plant injury for exposure to chlorine is similar to that by ozone or sulfur dioxide in many species (NAS, 1976).

Transportation Hazards

The primary risks associated with the transportation of chlorine are:

1. Human exposure to liquid chlorine
2. Human exposure, both occupational and public, to gaseous chlorine
3. Human injuries incurred without contact with chlorine

The quantification of risks associated with the transportation and handling of chlorine is made difficult by the variety of transportation modes available,and the wide range in sizes of shipping containers. Nevertheless, government regulations associated with transporting dangerous chemicals such as chlorine dictate an adequate data base for the risk analysis project.

Hazards of Process Reaction Products

The reaction products of the chlorination process that are hazardous are soluble organic and inorganic species. As a result the hazards are almost exclusively realized in the aquatic environment receiving the disinfected wastewater and in subsequent uses of those receiving waters.

Expected Reaction Products--

An assessment of risk associated with chlorine reaction products should identify and analyze all compounds formed or those whose concentrations increase during the disinfection process. However, the number of potential reaction products is large, so a limited number of compounds formed during the disinfection process were selected for risk assessment. Selection was based on reported effluent concentrations, toxicity, and the availability of published data.

Although several studies have identified a large number of chlorinated hydrocarbons in chlorinated secondary wastewater effluent (Glaze, 1975; Glaze, 1973; Jolley, 1975; Jolley, 1979; Environmental Protection Agency, 1979), selection of a small number of representative or model compounds for the risk analysis project has been difficult because of limitations within the published data bases.

Studies on chlorination reaction products are typically based on laboratory conditions which in turn often employ high chlorine doses and/or long contact times , neither of which are realistic when compared to normal treatment plant operating conditions. The daily, weekly, and seasonal variations in plant operating conditions add further complications in applying laboratory data to field conditions. Additionally, most studies used analytical techniques which were not effective in detecting nonvolatile compounds. Only recently has there been much effort placed on the difficult problem of separating and identifying nonvolatile chlorinated hydrocarbons (Jolley, 1979). Although a large number of chlorinated hydrocarbons have been separated in chlorinated wastewater effluent, not all have been unambiguously identified. A pilot study by the U.S. Environmental Protection Agency is studying chlorinated effluents under actual field conditions; however, the study is focusing on priority pollutants and does not attempt to identify all chlorinated hydrocarbons (Environmental Protection Agency, 1979a). Furthermore, toxicity data are lacking for most chlorinated hydrocarbons thus far identified in wastewater effluents.

The compounds selected for risk analysis are:

1. chlorine residuals
   (e.g. total residual chlorine (TRC)),
2. chlorinated aliphatic hydrocarbons
   (e.g. chloroform, trichloroethylene, and tetrachloroethylene), and
3. chlorinated aromatic hydrocarbons
   (e.g. dichlorobenzene, chlorophenol, and 5-chlorouracil).

Total residual chlorine was chosen because its concentration in chlorinated wastewater effluents is orders of magnitude higher than the chlorinated hydrocarbons (mg/L range versus ug/l range), and it is known to be toxic to aquatic life (Ward, 1978). Compounds in the second category represent low molecular weight, volatile chlorinated hydrocarbons. Although there is some evidence to suggest chlorination actually lowers the chloroform concentration in wastewater effluents (Environmental Protection Agency, 1979a), chloroform was chosen because of its relatively high concentration in chlorinated wastewater effluents. Tri- and tetrachloroethylene were chosen as representative volatile chlorinated hydrocarbons for which there was a reasonable amount of published toxicity information. Although other volatile compounds could have been chosen, most have very limited published toxicity information. The selection of chlorinated nonvolatiles given within the third category was more difficult due to very limited published information. Although the nonvolatiles seem to be present at the low parts-per-billion level, some are quite toxic and may represent significant risk to both humans and aquatic life. The choice of chlorobenzenes and chlorophenols was suggested by the availability of published toxicity data, and 5-chlorouracil was chosen because other investigators have selected this compound as a model for compounds typically found in chlorinated effluents (Gehrs, 1974).

Hazards to Freshwater Fish and Invertebrates--

Chlorinated wastewater effluents containing a variety of organochlorine compounds, total residual chlorine, and other inorganic and organic molecules, may have acute (short-term) toxic effects on aquatic organisms. Acute toxicity is generally caused by high concentrations of a given chemical during short-term exposures. The effects of these exposures are manifest immediately and often subside when the exposure is eliminated. In wastewater treatment plants acute exposures to high levels of toxic chemicals would generally coincide with opening a new plant, starting up after a shutdown, overchlorination during low wastewater flow or periods of low receiving stream flow.

Reactions to acute exposures of this nature could result in fish kills and death of invertebrates in extreme cases or slight physiological alterations in mild cases.

It is important to note, however, that acute toxicity may not always be caused by high concentrations of chemicals in the effluent. It is possible that highly toxic chemicals may exert their effects at low levels over a short time span.

Acute toxic effects range in severity from slight, reversible physiological perturbations to death. High levels of toxic chemicals are generally associated with acute toxicity, although the differential toxicity of chemicals precludes posing this as an inviolable rule. It may be more appropriate to talk of immediate effects rather than acute toxicity. Thus two types of immediate effects can be identified:

1. those caused by low levels of a chemical or chemicals, and

2. those caused by high levels.

Chronic toxicity includes effects which generally become manifest after considerable time. Induction of tumors is a classic example of a long-term or chronic effect. Concern for chronic effects is generally centered on exposure to low levels of pollutants over long periods. However, short-term exposures may also have long-term effects, i.e., a single exposure may not have an acute effect but may become manifest after a considerable period of time after the exposure. Chronic toxicity is generally discussed in reference to chemicals to which organisms are exposed for long-time periods. The effects of such exposures are not immediately apparent in most cases, but instead become manifest years from the commencement of exposure. Short-term exposures may also have a long-term, or delayed effect. Therefore, it is best to refer to delayed effects from both long-term and short-term exposure, rather than to use the term "chronic toxicity".

Exposure to chemicals in the long- or short-term may have a number of effects on aquatic organisms:

1. Alteration of normal physiological processes,

2. Induction of genetic mutations (mutagenesis),

3. Induction of cancer (carcinogenesis),

4. Induction of defects in offspring (teratogenesis),

5. Reproductive impairment of sexually mature individuals,

6. Decreased survival of eggs, embryos, and other life forms, and

7. Death.

These effects can drastically alter population size and may result in serious upsets in the food web. For example, loss of one species may result in subsequent losses in other species which are dependent on the first as a food source. These indirect effects are important in determining the overall impact of wastewater chlorination on aquatic organisms. This risk assessment focuses on the direct toxic effects of chemicals on fish, invertebrates and some aquatic plant-life (notably algae). It should be emphasized, however, that indirect effects (e.g., interruption of the food chain) may have serious consequences for aquatic organisms.

An additional consideration in assessing the impact of wastewater chlorination is the possibility that a chemical at a given concentration may not affect adults but may be lethal to eggs or fingerlings. This age-related differential sensitivity is an important factor to be analyzed in setting safe standards for aquatic pollutants.

Behavioral changes, while somewhat ignored by researchers and difficult to measure, could potentially occur in aquatic organisms exposed to pollutants. Reproductive behavior and migration, for instance, might be altered, thus affecting population size as well as survival. Crowding behavior and avoidance behavior (i.e., avoiding chlorinated water) have been reported in the literature. Their significance, however, to the overall stability of the aquatic ecosystem is not clear.

Hazards to Humans--

Humans consume surface water that, in some instances, contains chlorinated organics and chlorine residuals. These chemicals come from water chlorination at water and wastewater treatment plants. Exposure to these chemical species may have toxic effects. Immediate effects could range from slight alterations of normal physiology to severe consequences such as death. In reality, however, acute toxic effects are unlikely to occur from consumption of water containing total chlorine residuals and chlorinated organics because levels are generally quite low. The concern for human health is generally focused on long-term effects such as mutagenicity and carcinogenicity. Concern for mutagenicity and carcinogenicity of water chlorination by-products seems fairly well justified from the scientific literature. The mutagenic potential of water chlorination by-products deserves careful attention because of the seriousness involved in the alteration of the human gene pool and because of the large population potentially at risk. Teratogenic effects may also be manifest in human populations exposed to water chlorination by-products.

Additional Complications--

Chemicals may interact in a synergistic manner causing biological effects to be amplified. Synergism must be considered at all times in risk assessments because of the potential seriousness of the amplified effect. Analysis of studies on individual chemicals may indicate that none has a significant effect in low concentrations. However, exposure to several chemicals together at low levels may give a biological response which is greater than the sum of the effects when each chemical is given alone. Synergism cannot be predicted and unless studies are carried out with combinations of chemicals at low levels, so conservatism is required in predicting the risk of wastewater disinfection, especially with regard to mutagenesis, teratogenesis, and carcinogenesis.

It is also possible that chemicals may antagonize one another, i.e., the effects cancel out. The literature rarely refers to this potential, but it is possible that chlorinated by-products may have an antagonistic effect. In this case, the response of two chemicals together is less than the sum of their responses when administered alone.

Humans and aquatic organisms may be exposed to waterborne chemicals from a variety of sources other than wastewater disinfection. These sources of exposure should be considered carefully when choosing a wastewater disinfection alternative. In other words, the risk of a wastewater disinfection alternative should be integrated into the total risk picture in such a way that the total exposure to harmful chemicals can be made. This is especially significant since synergism and antagonism may occur with other environmental chemicals; that is, water chlorination by-products may synergize or antagonize with chemicals which enter an organism from air, food, medicine, etc.

An additional concern about the risks associated with wastewater disinfection reaction products is bioaccumulation. Bioaccumulation or biological magnification defines a phenomenon that occurs in food chains. Certain chemicals (e.g., DDT, chlorophenols, mercury) found in aqueous environments in low concentrations tend to accumulate in organisms. As one ascends the food chain, levels of these chemicals in the organisms increase. At the top of the food chain, concentrations are the highest and are often many thousands of times higher than in the aqueous environment. Thus, seemingly low levels of a chemical in water become hazardous since the organisms higher in the food chain have accumulated the chemical. Large concentrations of a chemical in the higher organisms may have a significant biological effect.

SEVERITY AND FREQUENCY OF IDENTIFIED HAZARDS

On-site Use Hazards

The goal for determining risk associated with the on-site use of chlorine as a disinfectant is to establish an accident frequency and the resulting severity rate for a variety of wastewater plant sizes. After reviewing a variety of data bases containing on-site accident information, the above goal was virtually impossible to attain. Thus, the on-site accident information for the use of all disinfectants has been listed as a data deficiency for this risk analysis project. This deficiency for on-site accident information has also been recognized by the Water Pollution Control Federation.

The data bases reviewed for on-site accident information will be discussed and shortcomings for the purpose of this risk analysis project will be highlighted. Finally, the data bases which allow an estimate of the risk associated with chlorine as a disinfectant will be reviewed, and a procedure for estimating on-site risk due to chlorine as a disinfectant will be developed.

United States Department of Labor--

Several departments, or bureaus, in the U.S. Department of Labor compile accident statistics for wastewater treatment plant workers. These include the Bureau of Labor Statistics, the Safety Programs Office and the Office of Management Data Systems, both of which are under the Occupational Safety and Health Administration (OSHA).

The annual accident statistics publications by the Bureau of Labor Statistics are quite general, do not break down the workers into a detailed four-digit Standard Industry Classification (SIC), and do not contain the causes of accidents. In these respects, these reports appear to be quite similar to those of the National Safety Council.

The Office of Management Data Systems and Statistical Analysis indicates four fatalities (three separate incidents) which occurred under SIC 4952 (sewerage systems) for the period 7/72 to 4/80 that were investigated by OSHA. Although the causes are not explicitly stated, standards from Title 29 of the Code of Federal Regulations are cited , and the severity of the citation is indicated. Further investigation may have revealed the causes for the accidents; however, most wastewater treatment plants are municipally owned and are not under OSHA requirements, thus limiting the OHSA data base severely.

In cooperation with state and local governments, the U.S. Department of Labor Bureau of Labor Statistics initiated the Supplementary Data System (SDS) to enhance the mechanism for collecting, coding, and analyzing statistical data concerning injuries and illnesses to workers. Although the SDS system can provide detailed accident information on wastewater treatment plant workers (or more precisely employees working on or for a wastewater treatment plant), the SDS data have proven to be unreliable for risk analysis purposes.

Although the exact reporting systems vary from state to state for SDS, the data are very detailed and suitable for determining the cause and severity of an injury for risk analysis purposes. Primary data from a typical SDS include:

1. Date of accident, date of claim, and/or claim number
2. Occupation of injured worker within a given SIC
3. SIC
4. Source of injury
5. Nature or type of injury
6. Severity of injury or extent of disability

Information required for the risk analysis project which is missing from the SDS include:

1. Treatment capacity of the wastewater treatment plant
2. Number of workers in SIC and plant or number in SIC state-wide
3. Number of employee-hours in SIC at plant or number in SIC statewide

Although SDS reports are open to the public, some states code information to prevent tracking accidents to the employer or individual treatment plant. When permissible, it may be possible to obtain some of this missing information by tracking accidents back to the specific treatment plant. In addition, some of the missing information could be obtained from other state agencies.

Missing information for SDS data was not pursued since, although the SDS is a good source of detailed information, it has some problems when trying to obtain accurate and reliable accident information for the risk analysis project. A brief review of these problems is given below:

1. There is apparently no federal requirement to belong to the SDS. In the western part of the United States, North Dakota, Kansas, Oklahoma, Louisiana, Texas, Nevada, and Illinois do not belong to the SDS while South Dakota is reportedly dropping out of the system.

2. Standardized reporting guidelines by the Department of Labor do not exist. Consequently, the type of information gathered, and reported, varies from state to state. Even though most states have computerized SDS data, obtaining data printouts is often time consuming, and the different coding systems and resulting different reporting formats dictate an explanation booklet accompany each state's data printout.

3. The SDS is typically coupled to the state workmen's compensation fund. Therefore, not all accidents are required to be reported. Accidents which result in less than a set number of lost workdays need not be reported. This "grace period" varies from state to state with a range of zero to seven days. A three to four day grace period is typical and determining the number of lost workdays is open to some subjective interpretation by employers. When trying to obtain reliable accident rates regardless of severity, this grace period is an obvious problem.

4. Data are open to bias from both the employer and state agency. Information for the SDS is compiled from reports submitted by employers concerning accidents of their employees. Employers may underestimate the type and severity of injury, may misrepresent the source of the injury, or may provide incomplete or ambiguous information (Colorado Division of Labor, 1978). State personnel who collect and compile the accident information may not be experts in classifying, or coding the information provided. The term NEC (Not Elsewhere Classified) is a category which is all too often encountered. In both cases, the reliability of the SDS data is open to question.

National Safety Council--

The National Safety Council (NSC) publishes Accident Facts, which is an annual publication that includes accident rates by SIC code. Unfortunately, wastewater treatment plant workers require a four digit SIC code and Accident Facts compiles information into two and three digit SIC codes. SIC grouping 495 is not one of the selected industrial groupings in Accident Facts.

Another publication by NSC, Work Injury and Illness Rates, includes a breakdown of injury and illness incidence rates by SIC code and industry. The 1980 edition is the first to include the 4952 SIC. The 1980 data covering the period 1977-1979 are obtained from only three reporting units, which are the minimum number of units required for publishing data. The usefulness of this limited data base is questionable.

Also contained in Work Injury and Illness Rates (National Safety Council, 1980) are data for SIC 4952 which the NSC collected in cooperation with the American Public Works Association. The data are collected from a substantially larger base and are summarized in Table 1 along with the NSC comparison data for transportation and public utilities as well as all industries.

TABLE 1. 1979 NSC ACCIDENT DATA

	reporting units	lost work-day cases	lost workdays
SIC 4952	156	8.21	115
SIC 4(transportation & public utilities)	1290	3.24	63
All Industries	8377	2.67	61

Note - lost workday cases and lost workdays are calculated using a base of 200,000 man-hours.

Water Pollution Control Federation--

The Water Pollution Control Federation (WPCF) compiles statistics on accidents occurring to wastewater treatment plant personnel. Results of the WPCF 1980 Safety Survey for accidents occurring in 1979 were published in the December 1980 issue of Deeds and Data (Water Pollution Control Federation, 1980). The WPCF data are based on 1422 survey responses which represent a substantial 7 to 10 percent of all wastewater works in the U.S. and Canada. Not only does the report use a substantial data base, but wastewater works are broken into collection and treatment facilities. The survey also includes the number of employees and man-hours for the reporting units. The WPCF data are summarized in Table 2.

The 1980 WPCF survey, as in all previous surveys, does not include information as to the cause or nature of the injury. The 1981 survey, which is to be compiled late in 1981, will include questions on the cause and type of injury. WPCF has recognized the need for more accident information on wastewater treatment plant personnel (Hadeed, 1981), and this detailed accident information would be most helpful in the risk analysis project.

TABLE 2. 1979 WPCF ACCIDENT DATA

plant size m^3 per day	man-hr per employee	injury frequency $/10^6$ man-hr	severity rate lost workdays $/10^6$ man-hr	fatalities $/10^6$ man-hr
<3790	1861	22.16	252.9	0
3790-9475	1985	38.99	210.9	0
9475-37900	1945	48.23	436.9	0
>37900	1958	61.95	749.3	0
average	1952	52.48	566.1	0

Notes
1. Data are for treatment plants only and do not include data for collections systems.
2. Plant size calculated assuming 379 liters per person per day.

American Water Works Association--

Although the American Water Works Association (AWWA) does not collect accident data for wastewater treatment plants, they do collect accident data for personnel at drinking water treatment plants. The AWWA data include injury frequency and severity rate for a parallel industry which uses chlorine as the disinfectant. The use of AWWA data appears to be valid since both water and wastewater treatment facilities are largely municipally owned, both industries use the same disinfectant and disinfection equipment, both industries show similar ranges in plant size, and both industries have about the same degree of automation. Furthermore, the AWWA data include information which is not available from the wastewater industry; namely, a breakdown of accident types. AWWA data are summarized in Tables 3

TABLE 3. SUMMARY OF 1979 AWWA ACCIDENT DATA

	units	Man-hr per employee	Injury Frequency $/10^6$ man-hr	Severity lost workdays $/10^6$ man-hr
Totals	2611	2021	35.19	1408

Notes
1. AWWA data include collection, treatment, and distribution phases of the water treatment industry for U.S. and Canada.

and 4 (American Water Works Association, 1979). The percentage figures shown in Table 4 are based on the frequency of occurrence, not on the severity measure, lost workdays.

TABLE 4. SUMMARY OF 1979 AWWA ACCIDENT DATA BY CATEGORY

INJURY	PERCENT
1. Sprain/strain in lifting, pulling, or pushing	24
2. Sprains/strains due to awkward position sudden twist or slip	16
3. Struck against stationary or moving objects	11
4. Struck by falling or flying objects	11
5. Falls on same level to working surface	7
6. Caught in, under, or between object	7
7. Falls to different level from platform, ladder, stairs, etc.	6
8. Contact with radiations, caustics, toxic, and noxious substances	4
9. Animal or insect bites	3
10. Rubbed or abraided	1
11. Contact with temperature extremes	1
12. Contact with electric current	0
13. Miscellaneous	10

For the purpose of comparison, accident types for disabling injuries from a state-wide industrial base for the State of Colorado are summarized in Table 5 (Colorado Division of Labor, 1978).

Before differences or similarities in the data can be examined, differences in the reporting systems must be considered. The National Safety Council uses the record keeping requirements found in the Occupational Safety and Health Act of 1970 (OSHA format) while the American Water Works Association and the Water Pollution Control Federation use the American National Standard Institute's (ANSI) method of recording and measuring work injury experience, ANSI Standard Z16.1-1967 which was reaffirmed in 1972 (ANSI Z16.1 format). Definitions for and differences between the two systems are noted by the National Safety Council (National Safety Council, 1980). The major differences are:

1. Z16.1 uses 1,000,000 man-hours while OSHA uses 200,000 man-hours to calculate incidence rates.

TABLE 5. 1977 COLORADO ACCIDENTS BY CATEGORY

Category	Percent
1. Overexertion	25.0
2. Struck by	19.9
3. Struck against	13.0
4. Fall on same level	9.7
5. Bodily reaction	6.2
6. Caught in, under, or between	7.5
7. Fall from elevation	7.5
8. Rubbed or abraded	2.4
9. Motor vehicle accidents	2.9
10. Contact with radiation or caustics	3.0
11. Contact with temperature extremes	2.2
12. Contact with electrical current, public transportation, NEC	0.6

2. Z16.1 measures ability to work whether scheduled to work or not (calendar days), while OSHA measures only scheduled workdays (five day work week) to calculate lost workdays.

3. OSHA has no schedule of time charges for deaths and permanent injury.

Since most wastewater treatment plants are municipally owned, OSHA record keeping is not required and the Z16.1 format is typically used.

Conclusions--

Data from the National Safety Council, the American Water Works Association, and the Water Pollution Contol Federation are useful for the purpose of risk analysis. The conclusions are summarized below:

1. Personnel employed at wastewater treatment plants have a poor overall safety record. The NSC SIC 4952 data compare quite well to that of the WPCF once the base man-hour difference is considered. These data point to an accident rate considerably above the average for all industries. The accident rate, in terms of total lost workdays for SIC 4952, is comparable to that for metal mining (National Safety Council, 1980) which is considered a rather hazardous occupation. Furthermore, the WPCF surveys (Hadeed, 1980) indicate that not only is the safety record poor, but that it has been declining in a long-term trend since 1967.

2. The WPCF survey indicates that the majority of accidents happens to employees involved with collection and distribution systems rather than the on-site treatment of wastewater. The AWWA also sees a larger portion of accidents off-site than on-site (Becker, 1981).

3. The WPCF data above indicate that in general the larger wastewater treatment plants have poorer safety records. This trend has been observed for some time for injury frequency rates, but not necessarily the severity rates over the same time frame (Hadeed, 1981).

4. Exposure to chlorine does not seem to be a major cause of accidents. In the AWWA accident category above, exposure to chlorine would fall in the category "contact with radiations, caustics, toxic and noxious substances". This comprises only 4 percent of the total accidents and puts it on a par with insect bites. However, the actual risk may be greater for chlorine exposure because of a greater severity factor. In comparing the AWWA category data to that for the State of Colorado, it appears that wastewater treatment accidents are very similar to those of all industries. The accidents caused by exposure to chlorine (chemicals) is not significantly higher in water treatment plants than in the general industry.

Risk Analysis Procedure--

The above data can be used to estimate the on-site accident rate for chlorine as a disinfectant. The AWWA category "contact with radiations, caustics, toxic and noxious substances" is almost entirely due to exposure to chlorine(Becker 1981). A minor contributor is exposure to organic solvents which are used in cleaning equipment. A conservative estimate would indicate that 4 percent of accidents are caused by exposure to the disinfectant chlorine.

The following procedure can be used to estimate the on-site accident rate for chlorine as a disinfectant.

1. Use WPCF severity rate data based on plant size

2. Use AWWA 4 percent figure to assign lost workdays from chlorine related accidents.

This simplistic procedure is based on three important assumptions:

1. The drinking water treatment plant data from AWWA is similar to that of wastewater treatment plant accident rates.

2. The severity (lost workdays) from exposure to chlorine is the same as the average severity from all types of accidents.

3. Chlorine accidents scale with plant size as all accidents.

Transportation Hazards

Introduction--

In this section, accident data from the Department of Transportation and U.S. census data on total chlorine movements are combined to yield output of the form:

$x(i)/y$ for railroad, truck (tank, 1 ton container or cylinders), or barge,

where,

$x(i)$ = deaths, injuries, dollars, or amount released;and

y = metric ton-kilometer.

This output, coupled with point-of-origin and point-of-destination information, will enable the reader to estimate the risk associated with the transportation of chlorine to a specific wastewater treatment plant. Point-of-origin information can be obtained from the map and tables of chlorine producers contained in this section and the appendices. A sample risk calculation for a particular treatment plant and details of the computations which were used to generate tables are also in this section.

Chlorine is transported as solid calcium hypochlorite, liquid sodium hypochlorite, or a compressed gas. However, almost all the chlorine used for wastewater disinfectation is transported as a compressed gas. For economic reasons, the percentage of total chlorine shipped for wastewater disinfection in the form of hypochlorite is neglible (Mitchell, R., Chlorine Institute).

Data Base--

Department of Transportation.--Since 1971 there has been a legal requirement that a report be filed with the Materials Transportation Bureau of DOT for every accident involving the transportation of a commodity. Since January 1971, the reports have included information on the number of deaths or injuries, and the amount of property damage; and since January 1976, the reports have also included the amount of material released. A computer print-out was acquired from the Materials Transportation Bureau covering the period from 1/71 - 12/80 (Morgan, 1980) and was used as an accident data base. The data for railroad, truck, and barge are summarized in Table 6. Note that in this table, the railroad accidents are shown with and without the major February 1978 rail accident in Youngstown, Florida.

TABLE 6. COMPILATION OF DOT ACCIDENT DATA, 1/71-12/80

	No. Accident Reports	Deaths	Injuries	Property Damage $	Amount Released kg
Railroad	72	8	247	1,111,498	1.402×10^5
Railroad Excl. Youngstown	71	0	87	22,498	0.993×10^5
Truck:					
Cylinders to 114 kg	14	0	60	8,003	574
0.911 Ton containers	4	0	15	23,550	245
Tanker Trucks	2	0	71	15,000	23
Barge	2	0	3	0	See note

Note - Information not available prior to 1976.

Bureau of Census.--Total chlorine movements by transportation mode and shipment weight can be obtained from the Commodity Transportation Survey of the U.S. Bureau of the Census. These data are compiled once every five years; the last published data are for the year 1972 (U.S. Bureau of the Census, 1972). Chlorine movements for 1972, in metric ton-kilometers, by transportation mode and by shipment weight are listed in Table 7. The breakdown of Census data by weight class and totals by transportation mode allowed total truck movements (15.0%) to be divided among tank trucks, 0.91 metric ton containers and 114 kg. cylinders. The results of this calculation can be obtained in the following manner. The 0.1% and 5% listed in Table 7 under 453 kg and 454-4539 kg, respectively, can be assigned to 114 kg cylinders and 0.91 metric ton cylinders, respectively. Tanker trucks were then assumed to carry the remaining 9.9% of the truck shipments. The result of this data analysis is shown in Table 8. A continued analysis of this sort shows that the values for truck shipments given in Table 8 are consistent with all of the data in Table 7.

The 1977 data was obtained prior to publication (R.Torene, 1981). Unfortunately, the unpublished 1977 data are less complete than the published 1972 data. Indeed, the quality of the 1977 data, particularly that of shipment weight, precludes the kind of analysis used to obtain Table 8. Since accident rates are

TABLE 7. PERCENT DISTRIBUTION OF CHLORINE SHIPPED BY TRANSPORTATION MODE AND BY SHIPMENT WEIGHT (TONMILES) FOR 1972

	%
A. Transportation Mode	
Rail	84.9
Truck (combines DOT motor carrier and private truck data)	15.0
Water	0.3
	100.2
B. Shipment Weight	
Under 454 kg	0.1
454 - 4,539 kg	5.0
4,540 - 13,619 kg	6.3
13,620 - 27,239 kg	6.1
27,640 - 40,859 kg	2.5
40,860 kg and over	80.2
	100.2

TABLE 8. BREAKDOWN OF CHLORINE SHIPMENTS BY TRANSPORTATION MODE AND CONTAINER

	Percentage (for 1972)	Annual Average metric ton-km (for 1971-1980)
Rail	84.9	1858
Truck:		
Cylinders to 114 kg	0.1	2.2
0.91 Metric Ton Cylinders	5.0	109
Tank Truck	9.9	219
Barge	0.3	6.4
	100.2	2194.6

calculated for truck shipments specific to container size, the 1972 data are used as a measure of the percentage of chlorine shipped by transportation mode and container for the period 1971-

1980. The average of the totals from the 1971 and 1977 census data, 2191 million ton-km, was used as a measure of the average yearly ton-km of chlorine shipped during this period.

Methodology and Calculation of Accident Rates--

The DOT data base over the period 1/71 - 1/80 (Table 6) was averaged to obtain an estimate of deaths, injuries, and property damage per year. The data base for the period 1/76 - 1/80 (Table 6) was averaged to obtain an estimate of chlorine released per year. The percentage breakdown by transportation mode and container shown in Table 8 was then used to calculate a corresponding breakdown by ton-km, assuming a yearly total of 2191 million ton-km. The results of this analysis, shown in Table 8, were used to normalize the accident data per ton-km. The accident rates calculated by this procedure are shown in Table 9.

TABLE 9. ACCIDENT RATES PER METRIC TON-KM

	Deaths	Injuries	Property Damage $	Chlorine Released kg
Railroad	4.3×10^{-10}	1.4×10^{-8}	6.0×10^{-5}	3.3×10^{-5}
Railroad excluding Youngstown	0	4.7×10^{-9}	1.2×10^{-6}	2.3×10^{-5}
Truck:				
Cylinders to 114 kg	0	2.7×10^{-6}	3.6×10^{-4}	1.2×10^{-4}
0.91 Metric Ton Containers	0	1.4×10^{-8}	2.15×10^{-5}	1.0×10^{-6}
Tanker Truck	0	3.2×10^{-8}	0.7×10^{-5}	4.6×10^{-8}
Barge	0	4.7×10^{-8}	0	See note

Note - information not available.

Table 9 shows that the accident rates for truck-transported cylinders are consistently higher than the other categories listed. This is likely due, in part, to the fact that a greater number of cylinders are needed to carry a given amount of chlorine. The deletion of the Youngstown accident from the railroad totals does not greatly alter the relative ordering of the accident rates in Table 9 except in the category of property damage per ton-km.

Location of Chlorine Producers--

Figure 1 (Chlorine Institute, 1980) shows the location of operating chlorine plants in the U.S. The chlorine producers and packaging plants are listed in the Appendices. These data can be used to obtain point-of-origin information in order to calculate specific accident rates to a particular wastewater treatment plant.

Sample Risk Calculation--

The risk associated with the transportation of chlorine to the Metropolitan Denver Sewage Disposal District No. 1 is used as a sample calculation. The chlorine used by Metro No. 1 is produced by National Lead of Salt Lake City, Utah (Puntenny, 1981). The chlorine is shipped the entire Salt Lake-Denver distance of 853 km by railroad (usually in 50 metric ton cars). Total chlorine usage was 374 metric tons in 1980 and 481 metric tons in 1981 (Puntenny,1981). Using the accident rates per ton-km in Table 9, the following risk factors are calculated for the 410,300 chlorine metric ton-km exposure of 1981:

Deaths	1.8×10^{-4} per year
Injuries	5.7×10^{-3} per year
Property damage	$25.00 per year
Chlorine released	14 kg per year

Process Reaction Products Hazards

Total Residual Chlorine--

Formation.--Chlorine applied to water in its elemental or hypochlorite form initially undergoes hydrolysis to form free available chlorine consisting of molecular chlorine, hypochlorous acid, and hypochlorite ion. The relative proportions of these free chlorine forms is pH dependent. At the pH of most waters the hypochlorous acid and hypochlorite ion will predominate.

In wastewater effluent and other types of waters, free chlorine reacts readily with ammonia to form monochloramine and dichloramine. The presence and concentrations of these combined forms depend on many conditions; chiefly pH, temperature, and the initial chlorine to ammonia-nitrogen ratio. Both free and combined chlorine may be present simultaneously. Chlorinated wastewater effluents and certain chlorinated industrial effluents normally contain only combined chlorine forms, and at the normal pH levels of such effluents the predominant species (and disinfectant) is monochloramine.

The oxidative products formed in chlorinated seawater are, for the most part, the same as in freshwater although the complex nature of sea water influences the relative abundance and type of chemical species found. The high chloride ion concentration in sea water influences the amounts of hypochorite, chlorite and

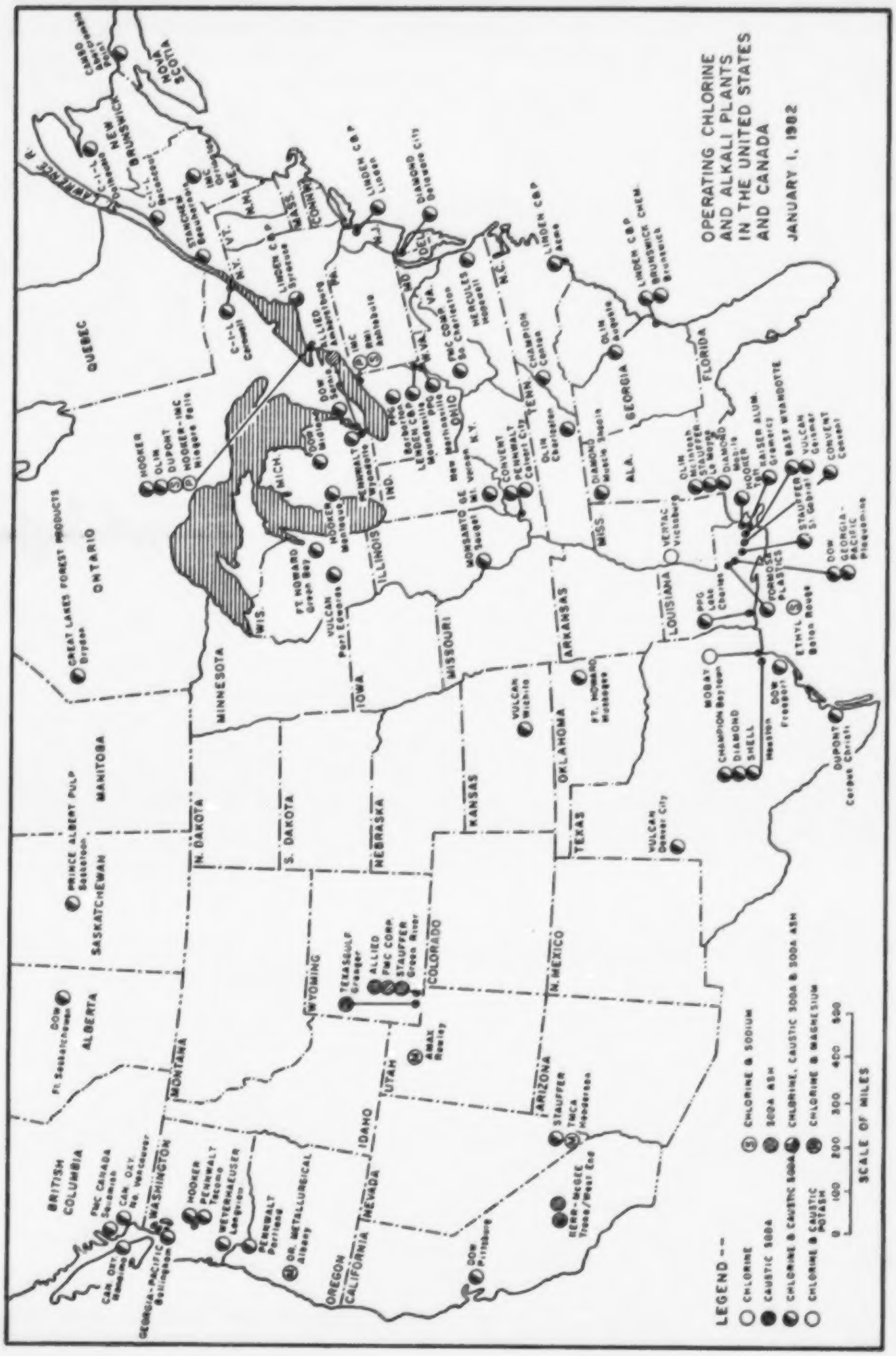

FIGURE 1. LOCATION MAP OF U.S. CHLORINE PRODUCERS
by permission of The Chlorine Institute

chlorate ions produced. The formation of chloramines is expected but concentration is variable and generally lower than in fresh water. Chlorine may react with other halogens (iodide, fluoride, bromide), but the principal reaction in seawater involves formation of hypobromous acid and hypobromite ion from bromine (Carpenter and Maculady, 1978). Bromamines, bromate, and bromine-chloride complexes may also form.

As with fresh water the nature and amount of chemical species found as products of chlorination of salt water will vary with the site-specific parameters of pH, temperature, reaction time, sunlight, and chemical composition of the water.

Persistence.--Because hypochlorous acid is a strong oxidizing agent, its stability in natural water is very low, especially at low pH. Hypochlorous acid rapidly oxidizes inorganic compounds and rates of reaction with organic compounds are generally slower. Generalizations on rates of loss are difficult to make because these rates depend on sunlight, depth of stream turbulence, temperature, pH, and type of reactants.

Monochloramine, being more stable, would be expected to persist for hours to days compared to the minutes to hours persistence for free chlorine. The bromine and bromamines formed in seawater are slightly weaker oxidizing agents, but they are less stable and and thus less persistent. The persistence of chloro-organics produced in chlorination is discussed in their respective sections below.

Use and Occurrence in the Environment.--Wastewater disinfection operations are presently governed by state water quality criteria in terms of meeting or exceeding specific levels of total or fecal coliforms in effluents. For example, California has adopted a bacteriological limitation based on the most probable number (MPN) of total coliform organisms. Eighty percent of the samples of effluent must contain an MPN less than 1000/100 ml (median of 240/100 ml) for coastal bathing waters, a median of 70/100 ml for shellfish growing areas, and a median of 23/100 ml for confined waters where human contact is possible with a dilution of at least 100 to 1. In order to achieve a total coliform count of 23/100 ml consistently in a well designed chlorination system, good quality secondary effluent would receive a chlorine dose in the range of 10-15 mg/L. The residual from such a system would be on the order of 2-4 mg/L. In a review of some 60 plants, White (1975) revealed that many of the plants operate in this range of dosage and residual. Systems that are inefficient may require dosages up to 25 mg/L with resulting residuals as high as 8 mg/L. Site-specific wastewater characteristics will have a great influence on the amount of chlorine needed to meet standards.

Residuals as high as those discussed above have a deleterious effect on aquatic life existing near the outfall. The most recent (1976) EPA criterion to protect aquatic life has set chlorine residuals at 0.2 mg/L (0.5 mg/L instantaneous maximum) free residual chlorine (FRC) for 2 hr/day, and to 0.002 to 0.010 mg/L total residual chlorine (TRC) for salmonid fish and marine and other fresh water organisms, respectively. Brungs (1976) has recommended a single criterion of 0.003 mg/L TRC for continuously exposed fresh water aquatic life.

Effects on Freshwater Vertebrates.--In order to determine the toxicity of chlorine to aquatic life, several factors must be considered. Chlorine is introduced at variable doses into freshwater ecosystems that are affected by a wide range of environmental conditions such as temperature, water quality, and species composition. The toxicity of the various forms of chlorine residuals to freshwater organisms is somewhat species specific. The generalization that cold water species (salmonids) are more sensitive to chlorine than warm water species may be an oversimplification in light of recent studies that have shown minnows and catfish to have median lethal concentration (LC-50) values close to those of the salmonids. At best, toxicity may be similar at genus level but generalizations above this level (i.e., family) are tenuous. There are conflicting data concerning toxicity with respect to the size of the fish. The differences may be due to test methods. Data on the effects of chlorine residuals on eggs, larvae and reproductive ability are also limited.

Avoidance behavior in fish to chlorinated effluents has been demonstrated in the laboratory and the field (Seegert, G.L. and Bogardus, R.B., 1980). The fact that fish kills related to chlorine are rare (Seegert, G.L. and Bogardus, R.B., 1980) and that lack of species numbers and diversity has been observed in rivers below waste treatment plants (Tsai, 1968, 1970), indicate fish can avoid low levels of chlorine residuals that are continuously discharged. The lowest concentrations avoided were 0.005, 0.035, and 0.050 mg/L for the mimic shiner, white bass, and bullhead minnow, respectively.

The metabolic functions of poikilothermic animals are directly tied to temperature and hence may be expected to influence the toxicity of chemicals in such organisms. Most of the studies to date indicate that the resistance of fish continuously exposed to chlorine is inversely related to temperature. The effects of temperature changes on toxicity are less a factor during long exposures than during short exposures.

Water quality characteristics play an important role in determining the responses of fish to chlorine. The pH of effluent water affects the relative proportions of the various chlorine species. The few studies done in this area suggest the role of pH in toxicity is probably related to the chemical species and not any direct effect on the organism. Many toxicity studies do not state water quality parameters and measurement of TRC does not reflect the toxicity of test solutions.

Effects on Freshwater Invertebrates.--Since invertebrates are part of the food chain in the aquatic ecosystem, the toxicity of chlorine residuals to these species is important. As with fish, chlorine residual toxicity varies greatly with species. The water flea, *Daphnia magna*, seems to be the most sensitive species with decreased reproduction at 0.002 mg/L and 100% mortality at 0.125 mg/L TRC (Brungs, 1973; Arthur, et al., 1974). The most resistant invertebrate species studied was the Oligochaete worm with an LC-50 of 91.0 mg/L (FRL) (Chung, S.L., 1960). More work is needed on invertebrate toxicology in order to judge the effects of the other water parameters previously discussed on these organisms.

Effects on Plants.--Aquatic plants act not only as shelter for fish and invertebrates, but also as substrates on which such organisms live. They are a food source, and they enrich the aquatic ecosystem by fixing carbon, thus increasing those foods necessary for energy expenditure. They also produce free oxygen required by all aerobic organisms. This fixation of carbon and the production of oxygen is done by the process of photosynthesis. The algae, *Chlorella pyrenoidosa*, the most sensitive species found, has been shown to experience a 50% decrease in growth at 0.18 mg/L (FRC, CRC) and a 43% mortality at 0.6 mg/L TRC (Kott and Edlis, 1969). Most of the species studied had decreased growth at a concentration of 2.0 mg/L TRC.

The literature dealing with the effects of TRC on freshwater organisms is massive. The key elements of each reported study reviewed in this study are summarized in tabular form in the Appendices. This mass of reported data is also summarized in a general format in Figures 2 and 3.

The reported data were first grouped according to the consequences observed as a result of chlorine exposure. Those consequences are avoidance behavior, mortality threshold, 50% mortality, and 100% mortality. These consequences are defined as:

Avoidance - detection of TRC by the organism, increase in environmental stress, altered behavior, depressed activity;

Mortality Threshold - increased mortality above that caused by natural cycles and events;

50% Mortality - corresponds to LC-50 data; significant mortality and stress; and

100% Mortality - absence of most aquatic organisms.

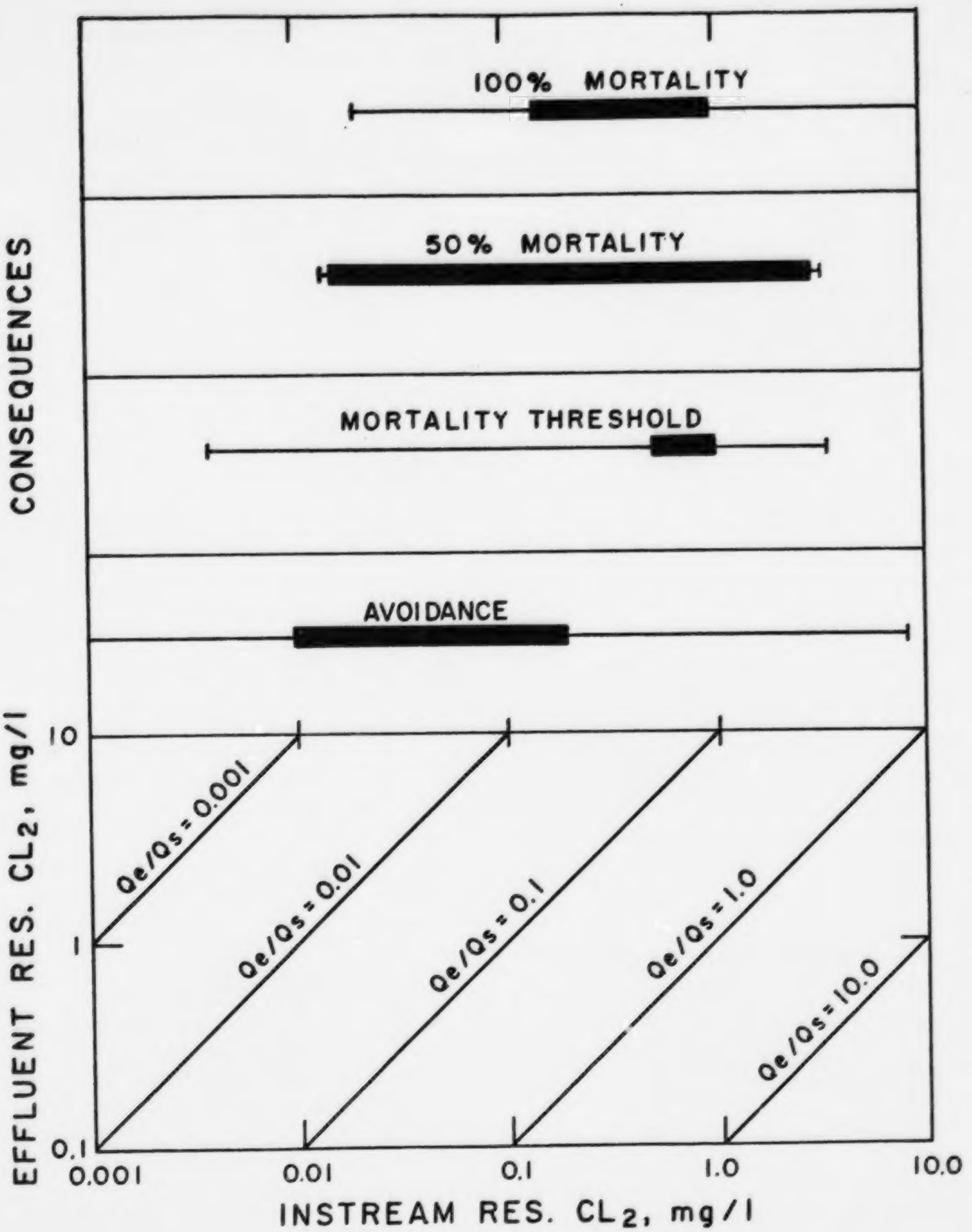

FIGURE 2. RESPONSE OF SELECTED FRESHWATER ORGANISMS TO TOTAL CHLORINE RESIDUAL

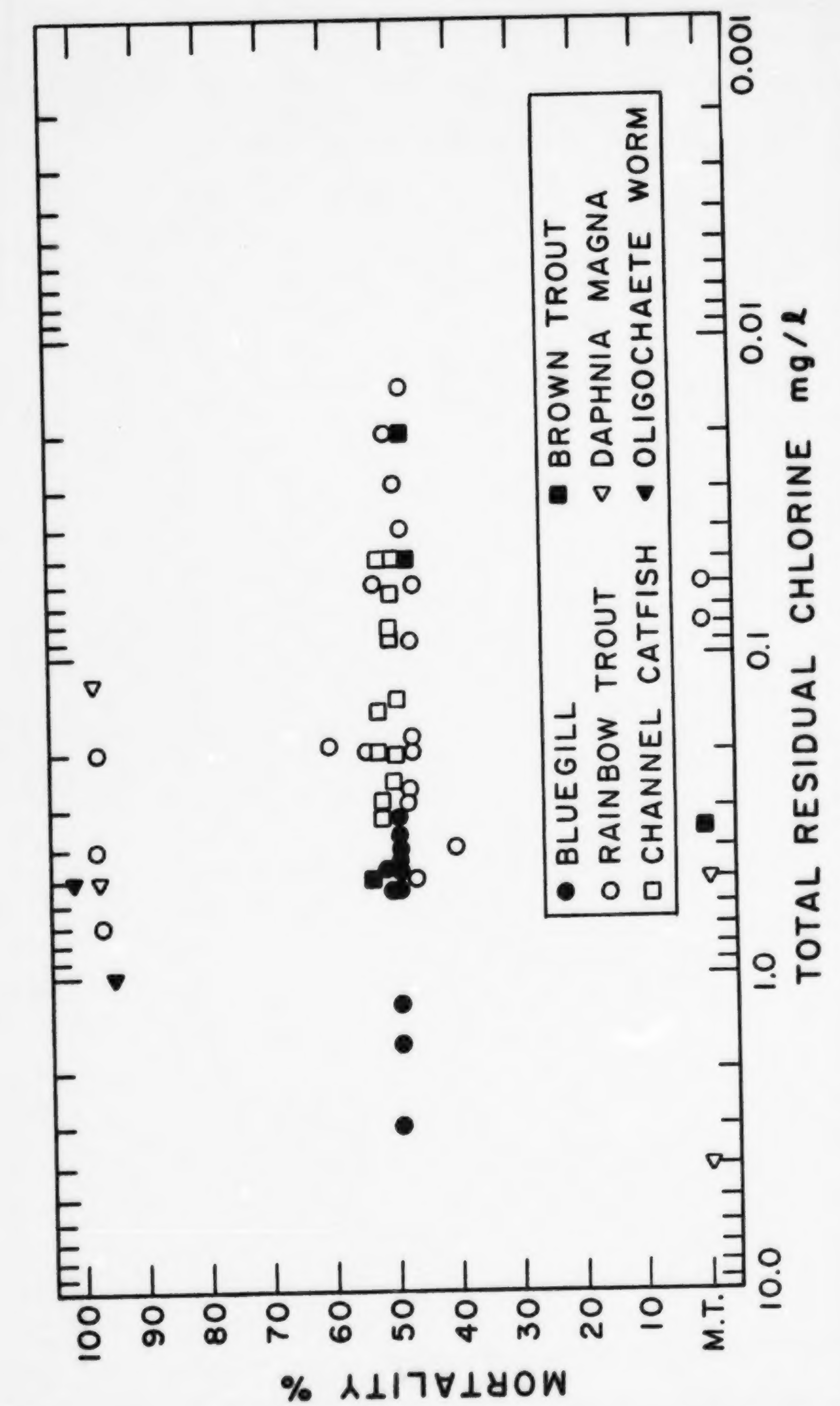

FIGURE 3. SUMMARY OF RESIDUAL CHLORINE EFFECTS ON FRESHWATER ORGANISMS

Each group of data was then plotted as a function of the reported chlorine residual. Figure 2 shows a sample plot for selected freshwater organisms. In Figure 3 the chlorine residual ranges containing many reported values are shown as heavy bars for each of the four consequences observed. The thin lines extending from each bar cover chlorine residual ranges with a few data reported. The ends of the thin lines represent the extreme values reported for each consequence. The overlap of the bars clearly illustrates the variability in the reported results. For example, the bulk of the data reported as mortality threshhold lie in the upper ranges of the data reported showing 50% and 100% mortality. In other words, an instream TRC of 1 mg/L might be at the mortality threshhold; or it might cause a 50 to 100% mortality.

To determine qualitative aquatic impacts using Figure 3, first estimate the effluent residual chlorine concentration (i.e.TRC). Then estimate effluent discharge (Qe = effluent discharge) and design stream flow (Qs = stream flow above discharge point) using the same units for both flow estimates. Next calculate Qe/Qs. Enter the graph at the lower lefthand scale using the estimated effluent TRC concentration. Move horizontally to the calculated Qe/Qs ratio line (if necessary interpolate between the lines shown in Figure 3). Then, move down vertically and read the in-stream residual chlorine (TRC) concentration. With the in-stream TRC concentration estimated then move up vertically to determine the possible effects on freshwater organisms.

Toxicity of Chlorine to Marine Vertebrates.--When comparing mortality data for several species of fish it was found that 96 hour LC-50 values were generally between 0.032 and 0.5 mg/L TRC (Gentile, 1974). A species of flounder was found to have 50% mortality at 2.5-10.0 mg/L over short time spans (0.345 mg/L). Although Atlantic Menhaden and Winter flounder eggs and larvae were unaffected at 0.5 (3 min) to 10.0 mg/L (20 min), several species, White Perch, Atlantic silverside, Fundulus heteroclitus, and Trinectes maculatus, when exposed to TRC concentrations from 0.03 - 0.08 mg/L (10 min), showed avoidance behavior (Gentile, 1974).

Toxicity of Chlorine to Marine Invertebrates.--The oyster Crassostrea vir. had a 46% decrease in ciliary movement when exposed to 0.2 mg/L TRC, and a pumping threshold at 1.0 mg/L (Galtsoff, 1962). The mussel Mytilus edulis was found to have unattached young at concentrations as low as 0.02-0.05 mg/L and the same species had 100% mortality at 1.0 mg/L over 15 days (James, 1967). Copepods of various species tested at different exposure times had mortalities from 22% to 90% at concentration ranges of 0.25 to 10 mg/L (Dressel, 1971; Coughlon and Davis, 1976; McLean, 1973). Barnacles had LC-90-100 values about 1.0 mg/L. Barnacle nauplii did not grow at 1.0 mg/L and the lethal concentrations for 12-62% of the test populations were 0.25 to 1.0 mg/L. Blue crab were shown to be affected by 0.10 mg/L (96 hour LC-50) while share crab had the same 96 hour LC-50 at 1.4 mg/L. The 96 hour LC-50 for sand shrimp was found to be 0.09 mg/L though the LC-55 for 10 minutes was 10.0 mg/L (Patrick and McLean, 1970).

Toxicity of Chlorine to Marine Plants.--The toxic effects to marine phytoplankton range from 50% decreased growth at 0.03 mg/L to 5-10 mg/L for Giant Kelp (Clendenning and North, 1959; Morgan and Stross, 1969). Most of the species tested were found to show 50% decreased growth between 0.10 to 1.0 mg/L chlorine.

Data Summaries.--The TRC data summaries in the Appendices also include reported data in each of the areas discussed above.

Chloroform--

Formation, definition, sources, and levels in the environment.--Chloroform ($CHCl_3$) is a low molecular weight, volatile hydrocarbon formed during the chlorination of wastewater as well as cooling and drinking water. Jolley, et al. (1973,1974,1975) report that approximately one percent of the chlorine applied to wastewater forms stable chloroorganics like chloroform. Unlike other chloroorganics discussed in this report, there are data on the levels of chloroform in the effluents of wastewater treatment plants. Concentrations generally range from 5 to 20 ug/L (NAS, 1978). Bellar, et al. (1974) report that the concentration of chloroform in wastewater prior to chlorination is 7.1 ug/L; after chlorination, the effluent chloroform concentration is 12.1 ug/L.

Chloroform enters surface waters (and thus drinking water) from a variety of sources other than wastewater treatment plants, including: 1) precipitation and 2) industry (paper mills, rubber manufacturers and chemical companies) (NAS, 1978). Effluent chloroform concentrations from industrial sources are usually higher than wastewater levels. For example, the NAS (1978) reported levels in paper mill effluents from 10 to 20,000 ug/L. Effluents from rubber manufacturers in Louisville and Calvert City, Kentucky ranged in concentration from 2,600 to 22,000 ug/L (NAS, 1978).

In the Region V organics survey of drinking water of 83 cities in the United States, the median concentration of chloroform was 20 ug/L; the maximum concentration was 366 ug/L (EPA, 1975). In the NORS survey of 80 municipal water sources, chloroform concentrations ranged from 0.1 to 311 ug/L; the median concentration was 21 ug/L. Mean concentrations in the atmosphere ranged from 0.045 to 4.0 ug/cubic meters.

Chloroform is formed in wastewater by the reaction of chlorine with natural and synthetic organic substances under alkaline conditions. There are numerous sources of organic matter, including humic substances, plant material, synthetics, and the end products of metabolic reactions of aquatic microflora.

The rates of these reactions are dependent on certain conditions, such as pH, temperature, and water quality. Generally, chloroform (and other trihalomethanes) production in chlorinated water is greatest at high temperatures (about 40° C) and neutral to high pH values (7 to 11), while the presence of ammonia reduces the formation of trihalomethanes.

Chloroform resists decomposition at ambient temperatures. Prolonged exposure to sunlight with or without air results in some decomposition, but the rate is not appreciable. Degradation of chloroform in water is accelerated by aeration and the presence of certain metals, such as iron (Hardie, 1964).

Bioconcentration of chloroform is fairly insignificant according to several published studies. Person and McConnell (1975) studied the levels of chloroform at various trophic levels and found no significant bioconcentration. The U.S. EPA (1978) reported a bioconcentration factor of 6 over 14 days in bluegills and a tissue half-life of less than one day.

Effects of Chloroform on Freshwater and Marine Organisms.--In freshwater fish several studies to determine the LC-50 have been carried out. In the Bluegill, Bentley, et al. (1975) report an LC-50 of 100 to 115 mg/L. Rainbow trout, on the other hand, are more sensitive to chloroform. Bentley, et al. (1975) report an LC-50 of 43.8 to 66.8 mg/L for adult rainbow trout. Clayberg (1971) reported 100% mortality in the orange-spotted sunfish at 106.9 to 152.7 mg/L. Apparently, chloroform is not highly toxic to freshwater fishes.

Data on the effects of chloroform to freshwater invertebrates are limited. The U.S. EPA report (1978) indicates that the mortality threshhold (i.e., concentration at which mortality commences) for *Daphnia magna* is between 1.8 to 3.6 mg/L. Furthermore, the LC-50 for *Daphnia magna* was reported to be 28.9 mg/L.

Several marine species have been studied in regard to their response to chloroform. Jones (1947) reported that anesthesia occurred within 90 minutes in Threespine Sticklebacks exposed to 207.6 mg/L. In the Ninespine Stickleback, Jones reported avoidance at 148.3 to 296.6 mg/L. The Pink Shrimp, *Peneus duorarum*, a marine species, was studied by Bentley, et al. (1975); they reported an LC-50 of 81.5 mg/L.

Effects of Chloroform on Humans.--There are three routes of entry in humans: 1) inhalation with absorption through the lungs, 2) ingestion with absorption through the gastrointestinal tract, and 3) dermal absorption. Pulmonary exposure to chloroform produces a rapid rise in blood levels of chloroform. Equilibrium is reached between blood and inspired air approximately 80 to 100 minutes from initial exposure (Lehman and Hasegaula, 1910). Absorption by the gastrointestinal tract is approximately 100 percent efficient (Fry, et al., 1972).

In mammals, including humans, ingestion of chloroform is not considered highly toxic. Ingestion of 30 to 100 ml of chloroform resulted in gastrointestinal disturbances accompanied by delirium and narcosis (vonOettingen, 1964). In humans, the mean lethal dosage is approximately 44 grams (Grosselin, et al., 1976).

Once in the blood, chloroform distributes throughout the body. The highest concentrations are found in peripheral nerves (Cornish, 1975). Interestingly, chloroform can be transported across the placenta. Fetal liver concentrations are higher than maternal liver concentrations (EPA, 1979).

Chloroform is partially metabolized by the liver and kidneys of mammals. Furthermore, it has been reported that chloroform is converted to CO_2 in the lung, but only to a small degree. The majority of absorbed chloroform is expired or excreted unchanged. Studies using ^{14}C-labelled chloroform in rats have shown after 18 hours 74% of the labelled chloroform was exhaled unchanged (vonOettingen, 1964). Fry, et al. (1972) report that 96% of the radioactively labelled chloroform given to adult humans was exhaled unchanged 8 hours after administration. Chiou (1975) reported that the half life of chloroform in the blood is 1.5 hours.

Chloroform mutagenicity was tested in bacterial systems. In the Ames Salmonella typhimurium test, chloroform did not prove mutagenic in strains TA1535 and TA1538. In Escherichia coli K-12, chloroform did not prove mutagenic (Uehleke, et al., 1976,1977).

Chloroform's teratogenic potential has been assessed in a number of studies, although most studies investigated inhalation. For example, Schwetz, et al. (1974) report that in rats 30 ppm inhaled for 7 hours per day on days 6 - 15 of gestation resulted in wavy ribs and delayed skull ossification, while inhalation of 100 ppm produced missing ribs, subcutaneous edema, imperforate anus, and delayed skull ossification. Thompson, et al. (1974) report fetal toxicity in rats and rabbits exposed orally to chloroform.

The International Agency for Research on Cancer (IARC) evaluated and published a review of several experiments which attempted to evaluate the potential carcinogenicity of chloroform. These experiments, including evaluations by the National Cancer Institute, were performed in mice, rats, and dogs with a variety of routes of administration. Doses ranged from 15 to 100 mg/kg/day and duration of exposure ranged from 8 weeks to 7.5 years (in dogs). The IARC concluded that chloroform is carcinogenic in mice (liver) and rats (kidney), and that chloroform presents a carcinogenic risk to man.

Epidemiologic surveys of the potential cancer risk of chloroform have been made. The IARC reviewed the study of Bomski, et al. (1967), but reported that the study was inadequate to draw any conclusions because of the small numbers used and the short follow-up. On the EPA's request, the National Research Council in 1978 reviewed 10 studies on the association between cancer and trihalomethane consumption in drinking water (EPA, 1979). The Council concluded that in most studies, the exposure and duration levels were inferred, and that there were inadequate controls and other invalidating factors. Thus, it is impossible to make a risk evaluation of chloroform in humans from this data.

The risk of developing cancer from the consumption of chlorofrom has been calculated by the National Academy of Sciences. Using a linear, non-threshhold extrapolation from animal data, the lifetime risk was estimated to be 1.5×10^{-7} to 17×10^{-7} per microgram of chloroform per liter. This means that 1.5 to 17 cases of cancer will occur in a population of 10,000,000 if the drinking water contains 1 ug/L chloroform. The EPA also calculated risk levels and found that if the level of chloroform is 1.21 ug/L, 1 case of cancer can be expected to occur in a population of one million. Recently, however, the EPA modified its calculation of risk.

Estimates of risk depend on accurate estimates of exposure. Both the EPA and NAS have estimated the annual exposure of humans to chloroform under a variety of conditions. While these values are equivocal, they may at least be used to estimate approximate cancer rates. The problem lies in determining which exposure model is most accurate and most typical. From Table 10, it is clear that annual exposure (in mg/yr) is the same in adults and children at each concentration exposure. For instance, at a minimum concentration exposure (0.0001 mg/L), annual uptake in adult men, adult women, and children is calculated to be about 0.037 to 0.088 mg/yr. At median concentration exposure, the uptake is estimated to be about 7.6 to 18 mg/yr and at maximum concentration exposure, 13 to 320 mg/yr. These estimates include chloroform uptake from tap water, as well as water-based drinks.

In Table 11, also from the NAS study, the estimated exposure to chloroform from all sources is given. At minimum concentration exposure (minimum levels in drinking water), fluids provide a minor portion of the total chloroform uptake; at typical and maximum exposure levels, however, fluids provide the largest

TABLE 10. CHLOROFORM UPTAKE FROM FLUIDS(NAS,1978)
mg/year (assuming 100% absorption)

Exposure Level mg/L	Fluid Source	Adult Man		Adult Woman		Child(5-14 yrs.)	
		Min. Intake	Max. Intake	Min. Intake	Max. Intake	Min. Intake	Max. Intake
0.0001 (min. level)	Tap water	0.016	0.027	0.016	0.027		
	All	0.037	0.088	0.037	0.088	0.036	0.061
0.021 (median level)	Tap water	3.44	5.59	3.44	5.59		
	All	7.57	18.4	7.67	18.4	7.67	12.8

proportion of the chloroform. From these data, it would appear that the range in total annual chloroform exposure is quite large -- from a minimum of about 0.6 mg/yr to well over 800 mg/yr. If this range is accurate, estimates of the cancer risk can be made to indicate the extremes. Furthermore, the relative contribution of drinking water to the cancer risk can be estimated.

TABLE 11. HUMAN UPTAKE OF CHLOROFORM
FROM ENVIRONMENTAL SOURCES (NAS, 1978a)
mg/year

Exposure Level	Source	Adult Man	Adult Woman	Child
Minimum	Fluid Intake	0.037	0.037	0.036
	Atmosphere	0.41	0.37	0.27
	Food	0.21	0.21	0.21
	Totals	0.66	0.62	0.52
Typical	Fluid Intake	14.90	10.70	10.70
	Atmosphere	5.20	4.70	3.40
	Food	2.17	2.17	2.17
	Totals	22.27	17.57	16.27
Maximum	Fluid Intake	321	321	223
	Atmosphere	474	434	310
	Food	16.4	16.4	16.4
	Totals	811	771	549

Table 12 shows estimates of uptake of chloroform from a variety of sources, as determined by the EPA. Adult uptake varies from a low of 3.14 mg/yr to a high of 563 mg/yr, a range less than that determined by the NAS.

TABLE 12. HUMAN UPTAKE OF CHLOROFORM FROM AIR, WATER AND FOOD (USEPA,1978b)

Exposure Level	Source	Uptake mg/year	Percent of Total Uptake
Maximum	Atmosphere	204	36
	Water	343	61
	Food	16	3
	Totals	563	100
Mean	Atmosphere	20	21
	Water	64	69
	Food	9	10
	Totals	93	100
Minimum	Atmosphere	0.41	13
	Water	0.73	23
	Food	2.00	64
	Totals	3.14	100

Trichloroethylene--

Formation, Definition, Sources and Levels in the Environment.--Trichloroethylene (1,1,2-trichloroethylene, C_2HCl_3) is a volatile chlorohydrocarbon formed during wastewater disinfection as well as drinking water chlorination (Bellar, et al., 1974). Little data is available on the production or occurrence of trichloroethylene in wastewater treatment plants. Bellar, et al. (1974) report that wastewater treatment plant influent may contain as much as 40 ug/l of trichloroethylene. This study also reported that chlorination increased the trichloroethylene level from 8.6 ug/l to 9.8 ug/l. Certainly, more work is necessary to evaluate the production of trichloroethylene from wastewater treatments plants. Trichloroethylene also enters surface waters (and therefore drinking water) from other sources such as precipitation and industry (IARC,1979).

The U.S. National Organics Monitoring Survey (EPA,1976) found that about one-fifth of the municipal water supplies tested did not contain trichloroethylene and that the detected levels were quite low (EPA, 1978b). Mean concentrations of trichloroethylene ranged between 1.3 to 21 ug/L (IARC, 1979).

Toxicity of Trichloroethylene to Freshwater and Marine Organisms--Trichloroethylene is toxic to freshwater fish; but, like tetrachloroethylene, its toxicity is low (Alexander, et al., 1978; Pearson and McConnell, 1975 and EPA, 1978b). The 96-hour LC-50 for the Fathead Minnow is 36.5 mg/L with static testing and 40.7 with flow-through testing (Alexander, et al., 1978). The 96-hour LC-50 for Bluegills is 44.7 mg/L under static testing conditions (Alexander, et al., 1978).

Little work has been directed toward the elucidation of the effect of trichloroethylene on freshwater invertebrates. Studies on *Daphnia magna* indicate that the 48-hour LC-50 is approximately 85.2 mg/L under flow-through testing conditions (EPA, 1978b). In life cycle tests, 10 mg/L had no effects (Alexander, et al., 1978). The unicellular alga, *Phaedactylum tricoinitum*, showed a 50 percent decrease in the uptake of radioactive carbon during photosynthesis at a concentration of 8.0 mg/L (McConnell and Pearson, 1975). The Dab, a saltwater fish, had a 96-hour LC-50 of 16 mg/L (McConnell and Pearson, 1975). In the Sheepshead Minnow, exposure to 2.0 mg/L of trichloroethylene produced erratic swimming, uncontrolled movement and loss of equilibrium.

Salt water invertebrates have also been tested, but in a rather limited manner. Grass Shrimp displayed erratic swimming, uncontrolled movement and loss of equilibrium after exposure to 2.0 mg/L of trichloroethylene (Borthwick, 1977). In barnacle nauplii the 48-hour LC-50 was 20 mg/L (McConnell and Pearson, 1975).

On the whole, there are not much data concerning the toxicity of trichloroethylene in aquatic ecosystems, which makes the construction of dose-response and risk assessments impossible. From the limited data above, however, it appears that the toxicity of trichloroethylene is low. Furthermore, because the levels of trichloroethylene are low in the effluents of wastewater treatment plants, acute toxic reactions in fish and other aquatic organisms below wastewater outfalls are unlikely. Further studies may show that low levels of trichloroethylene have chronic effects such as teratogenicity, mutagenicity and carcinogenicity.

Effects on Humans.--Studies of the effects of trichloroethylene on humans are limited. Most studies are of occupationally exposed individuals who were subjected to rather high concentrations of the chemical in the air. Most of these studies indicate an effect on the central nervous system in workers exposed to trichloroethylene; reported findings include mild fatigue, decrease in psychomotor function, decrease in performance ability, and headache (Stuart, et al., 1970; Stopps and McLaughlin, 1976; Salvini, 1971; Nomiyama and Nomiyama, 1971).

No data were available on the teratogenicity of trichloroethylene in humans, and animal studies show little, if any, effect. Schwetz, et al. (1975) tested mice and rats for possible teratogenic effects, but their results were inconclusive.

No human data are available for assessment of the mutagenic potential of trichloroethylene. Mutation rate was increased by the administration of trichloroethylene to a number of bacterial strains including *E. coli* and *Salmonella typhimurium* (IARC, 1979). Similar findings were obtained in the yeast, *Saccharomyces cerevisiae*; however, impurities in the technical grade trichloroethylene used in the these experiments now have been shown to be mutagenic agents (IARC, 1979).

Only one study of the possible carcinogenic potential of trichloroethylene in humans was found. The incidence of cancer mortality was studied in a population of 518 workers occupationally exposed to low levels of trichloroethylene. Exposure was detected by urine analysis. No excess in cancer mortality was noted in this study; however, the limited size of the study group precluded detection of cancers such as liver cancer which are found in a low incidence (Axelson, et al., 1978 in IARC, 1979).

Studies in laboratory animals indicate that trichloroethylene may be carcinogenic. For instance, trichloroethylene induces transmutation in Fisher rat embryo cells *in vitro*, a study system used for the identification of carcinogens. Second, a 1976 NCI study indicated an increase in the incidence of hepatocellular carcinoma in mice; Osborne Mendel rats, however, showed no increase in cancer (IARC, 1979).

Tetrachloroethylene--

Formation, definition, sources and levels in the environment.--Tetrachloroethylene (C_2Cl_4) is a volatile chlorohydrocarbon (also known as perchloroethylene) formed in wastewater disinfection (Bellar, et al., 1974). Unfortunately, there is little data on the level of tetrachloroethylene found in wastewater treatment plant effluents. Bellar, et al. (1974) reported that chlorination of wastewater slightly increases the concentration of tetrachloroethylene with final effluent concentrations of about 4.2 ug/L.

Tetrachloroethylene enters surface waters (and thus drinking water) from a variety of industrial sources other than wastewater treatment plants (Shakelford and Keith, 1970).

Generally, levels of tetrachloroethylene are quite low in surface waters. The Safe Drinking Water Commission (1977) found that 8 of 10 samples from different water supplies contained tetrachloroethylene, but only in low concentrations, ranging from 0.07 to 0.46 ug/L (IARC,1979). In the U.S. EPA's National Organics Monitoring Survey (EPA,1976), tetrachloroethylene was detected in 9 of 105 drinking waters sampled. Again, levels were quite low; for instance, the mean concentration of the nine samples was 0.81 ug/L and the range was 0.2 to 31 ug/L (U.S. EPA, 1978).

Effects of Tetrachloroethylene on Aquatic Organisms.--Tetrachloroethylene is toxic to freshwater fish, but the toxicity is relatively low compared to total residual chlorine. In the Bluegill, the 96-hour LC-50 is approximately 12.9 mg/L (EPA, 1978). In the Fathead Minnow, the 96-hour LC-50 is approximately 18.4 to 21.4 mg/L (Alexander, et al., 1978). The Bluegill and Fathead Minnow are both warm water species. Unfortunately, no LC-50 data are available for cold water species. The 96-hour LC-50 for the Sheepshead Minnow was 29.4 to 52.2 mg/L (EPA, 1978), slightly higher than the LC-50 for freshwater fishes.

Freshwater invertebrates are also affected by tetrachloroethylene. Unfortunately, evidence is sparse. The 48-hour LC-50 for *Daphnia magna* is 17.7 mg/L (EPA, 1978).

Unicellular organisms such as algae are affected by tetrachlorethylene; however, in these organisms its toxicity is relatively low. In several species of algae, EC(50) (effective concentration) ranged from 10,000 to over 800,000 mg/L (Pearson and McConnell, 1975; EPA, 1978).

In summary, because of the lack of data on the toxicity of tetrachloroethylene in fishes, a dose-response curve cannot be constructed. It does appear evident though, at least for the species discussed above, that since tetrachloroethylene concentrations in the effluents of wastewater treatment plants are low, acute toxic reactions in stream fish below the outfall are unlikely. Further studies may show that low levels have chronic effects such as mutagenicity, carcinogenicity, and teratogenicity.

Effects of Tetrachloroethylene on Humans.--The chief effects of tetrachloroethylene in humans are central nervous system depression, hepatotoxicity and neural disorders. Most studies of these effects, however, examine the inhalation of fairly high concentrations of tetrachloroethylene.

Only two systematic studies of human carcinogenicity were found. These studies indicate that tetrachloroethylene may be carcinogenic. Blair, et al. (1978) mentioned a clinical report of five cases of chronic symptomatic leukemia in a family that operated a dry cleaning business. Blair, et al. (1979) studied death certificates from deceased laundry and dry cleaning employees who had been exposed to tetrachloroethylene, trichloroethylene and chloroform. He observed an excess of cancer in certain loci: cervix, lungs and skin. Liver cancer and leukemia exhibited a slight excess. Blair's study was inconclusive due to the small sample size.

Animal carcinogenicity studies show mixed results. B6C3 mice orally exposed to large doses of tetrachloroethylene for 78 weeks (5 days per week) displayed an increase in hepatic carcinoma (NCI, 1977). Rats, similarly exposed, did not have an increase in cancer (IARC, 1979).

The mutagenicity of tetrachloroethylene, like its carcinogenicity, is not resolved. Cerna and Kypenova (1977) tested *Salmonella typhimurium* (TA 100) and found some mutagenicity; however, in mice tetrachloroethylene did not appear to be mutagenic (Cerna and Kypenova, 1977). Greim, et al. (1975) failed to elicit a mutagenic response in *E. coli* treated with tetrachloroethylene.

Chlorophenols--

Formation, Definition, Sources and Levels in the Environment.--The chlorophenols are nonvolatile chlorohydrocarbons which are formed during the chlorination of wastewater as well as drinking water (Jolley, 1973; Jolley, et al., 1975; Glaze and Henderson, 1975). Chlorine reacts readily with phenol in aqueous solutions over a wide range of pH values (Carlson and Caple, 1977). Levels in the effluents from wastewater chlorination are in the .001 mg/L range.

Burtschell, et al. (1959) proposed a scheme for the mechanism of phenol chlorination in water. Monochlorophenols (2- and 4-chlorophenol) are formed first; further chlorination of these species produces 2,6- and 2,4-dichlorophenol. After 18 hours of reaction, the products consisted of: 1) less than 5 percent monochlorophenol, 2) 25 percent 2,6-dichlorophenol, 3) 20 percent 2,4-dichlorophenol and 4) 40 to 50 percent 2,4,6-trichlorophenol.

Like other chlorohydrocarbons discussed in this report, chlorophenols enter surface waters from a variety of sources other than wastewater treatment facilities. Industrial processes are the dominant source of these pollutants (EPA, 1976; IJC, 1980).

Effects of Chlorophenols on Aquatic Organisms.--A substantial amount of data have been reported on the effects of chlorophenols on aquatic organisms. Key elements of these reports are summarized in tabular form in the Appendices.

The toxicity of the chlorinated phenols to aquatic organisms varies with the degree of ring substitution as well as the position of the chlorine atoms on the ring. In general, toxicity increases with increasing substitution. The more toxic multichlorinated products may come from industrial effluents rather than wastewater and drinking water chlorination (IJC, 1980).

Among the freshwater fish, Bluegills (*Lepomis macrochirus*) are slightly more sensitive than Fathead Minnows (*Pimephales promelas*), Goldfish (*Carassius auratus*) or Guppies (*Poecillia reticulata*). LC-50 (96 hour) for Bluegills range from 0.140 to 10.0 mg/L (Table 13).

In Fathead Minnows toxicity (96-hour LC-50) varies, depending on the degree of substitution, from 0.03 to 20.17 mg/L. Forty-eight hour LC-50's in Rainbow Trout (*Salmo gairdnerii*) range from 0.023 to 10.0 mg/L.

The toxicity of chlorophenols to aquatic invertebrates has been fairly well documented (Jolley, 1973; Jolley, et al., 1975; Glaze and Henderson, 1975). In *Daphnia magna*, toxicity ranges from 0.29 to 7.43 mg/L; again, the variation in toxicity (measured as 96-hour LC-50) results from the degree of substitution. Altogether toxicity among fishes and invertebrates (i.e., *Daphnia*) is fairly similar. The response of freshwater algae and duckweed to a variety of chlorophenols has been studied fairly extensively. There is a wide range in the effective concentrations resulting from differences in chemical species as well as plant species. It appears that polychlorinated phenols are most toxic (Huang and Gloyna, 1967 and 1968; EPA, 1978; Blackman, 1955; Erickson and Freeman, 1979).

Limited data are available on the toxicity of chlorophenols to marine fishes and invertebrates. Several studies of the Sheepshead Minnow show that toxicity (measured by the 96-hour LC-50) range from 1.66 to 5.35 mg/L (EPA,1978).

Mysid shrimp appear to be less sensitive to chlorinated phenols. A considerable amount of data is available regarding the effects of chlorinated phenols on saltwater algae. Again depending on the end point under consideration, chemical species, and algal species, the 96-hour LC-50 are present in a wide range from 0.25 to approximately 8 mg/L (EPA, 1978; Erickson and Freeman, 1979).

In summary, it would appear that chlorophenols are toxic, but not as much as residual chlorine. If the available data are representative of wastewater disinfection facilities, then stream concentrations would be expected to fall well below toxic levels.

However, significant bioconcentration can result in toxicity which might not be predictable from the acute toxicity data previously reviewed in this section. Bioconcentration factors in fish for various chlorophenols range from 8 to 240 with the more substituted compounds having higher factors (EPA, 1980).

Effects of Chlorophenols in Humans.--No data could be found on the levels of chlorophenols in ambient air. Toxicity of chlorophenols in mammals is well known. 2-chlorophenol, as well as 3- and 4-chlorophenol, is an "uncoupler" of oxidative phosphorylation (Mitsuda, et al., 1963). In addition, these compounds are known to cause convulsions (Farquharson, et al., 1958; Angel and Roger, 1972). Apparently, gastric acidity allows chlorophenols to be converted to more soluble forms which are readily absorbed by the intestines.

In rats, 2-chlorophenol produces symptoms including restlessness, increased respiration rate, followed by weakness, tremors, convulsions, dyspnea, coma and death (Farquharson, et al., 1958). At high concentrations, 2-chlorophenol produced fatty degeneration of the liver, renal granular dystrophy, and degeneration of intestinal mucosa (Bubnov, et al., 1969).

Acute poisoning with 2,4,5-trichlorophenol impairs physical activity by inducing motor weakness and convulsion; however, the trichlorophenols tend to produce fewer and milder convulsions than the monochlorophenols (Farquharson, et al., 1958; Deichmann, 1943). Weinbeck and Garbus (1965), Parker (1958) and Mitsuda, et al. (1963) report that, like the monochlorophenols, trichlorophenols uncouple oxidative phosphorylation, but only weakly.

A number of reports have disucssed the carcinogenicity of the chlorophenols. Table 13 summarizes the results of work reported in this area.

A study of tumor promotions in mice by chlorophenols was done by Boutwell and Bosch (1959). 20% solutions were applied weekly to female Sutter mice (2-3 months old) for 15 weeks. This followed the application of a tumor initiator, DMBA (9,10-dimethyl -1,2-benzanthracene). Two monochlorophenols (2- and 3-) were found to promote papillomas. 4-chlorophenol was not tested. The trichlorophenols (2,4,6- and 2,4,5-) were tested in the same manner with 2,4,5- increasing the incidence of papillomas in DMBA pretreated mice. A bioassay was conducted by the NCI on 2,4,6-trichlorophenol in the feed of F344 rats and B6C3F1 mice. The NCI concluded from these studies that 2,4,6-trichlorophenol was carcinogenic in male rats (F344) (producing lymphomas or leukemias). In addition, this compound was also found to be carcinogenic in both sexes of B6C3F1 mice (producing hepatocellular carcinomas and adenomas). It remains to be seen at this writing, whether 4-chlorophenol, 2,3,6-trichlorophenol, and 2,3,4,6--tetrachlorophenol are carcinogenic.

TABLE 13. CARCINOGENIC ASSESSMENT OF CHLOROPHENOLS IN LABORATORY SPECIES

Compound	Results
2-chlorophenol	possible promoter
3-chlorophenol	possible promoter
4-chlorophenol	not known
2,3,6-trichlorophenol	not known
2,4,5-trichlorophenol	possible promoter
2,4,6-trichlorophenol	possible carcinogen in rats and mice
2,3,4,5-tetrachlorophenol	not known

The EPA has calculated cancer risk level for only one of the chlorophenols, 2,4,6-trichlorophenol. The probability is 0.5 that a 2,4,6-trichlorophenol concentration of 3.6 ug/l will cause 1 additional case of cancer in a population of 1 million people.

Monochlorophenols have not been tested for mutagenicity. Two test systems have been used to test the mutagenicity of trichlorophenols. Fahrig, et al. (1978) found that 400 mg of 2,4,6-trichlorophenol increased the mutation rate in *Saccharomyces cerevisiae*. Rasanen, et al. (1977) reported that several polychlorinated phenols (2,3,5-, 2,3,4,6-, 2,3,6-, 2,4,5- and 2,4,6-) were not mutagenic when tested via the Ames test.

Dichlorobenzenes--

Formation, Definition, Sources and Levels in the Environment.--The dichlorobenzenes (DCBs) are chlorinated benzenes produced during the chlorination of wastewater (Glaze and Henderson, 1975). Three isomers of dichlorobenzene exist: 1,2-dichlorobenzene, 1,3-dichlorobenzene , and 1,4-dichlorobenzene. The DCBs are readily soluble in fats (Windholz, 1976) and are relatively volatile (Jordan, 1954; Kirk and Othmer, 1963; Varschueren, 1977). Glaze and Henderson (1975) reported that levels of DCBs in effluents from wastewater treatment plants were about 10 ug/L.

DCBs have been detected in rivers, groundwater, industrial and municipal wastewater, air, and soil. Ware and Ware (1977) reported that the average levels of 1,2-DCB in industrial wastewater were 2 mg/L. Glaze, et al. (1976) reported that DCB can be produced during the chlorination of municipal wastewater.

Glaze and Henderson (1975) reported that both 1,2-DCB amd 1,4-DCB concentrations were about 10 ug/L in chlorinated municipal wastewater effluents.

The EPA (1975) reported detecting low concentrations of DCBs in municipal drinking water samples. For 1,2-DCB, 1,3-DCB and 1,4-DCB reported concentrations were 1, <3, and 1 ug/L, respectively.

Effects of Dichlorobenzene on Aquatic Organisms.--Dawson (1977) found the 96-hour LC-50 for 1,2-DCB in Bluegills was 27.0 mg/L, while the EPA reported 96-hour LC-50s of 4.28 and 5.59 mg/L. The large difference in the reported LC-50 values may result from different methods of chemical dispersion used by investigators. In the Fathead Minnow, the EPA reported the 96-hour LC-50 for 1,4-DCB to be 4.0 mg/L. Rainbow trout were slightly more sensitive than Bluegills and Fathead Minnows according to the 1980 EPA study in which LC-50s for 1,2-DCB and 1,4-DCB were reported as 1.58 and 1.12 mg/L, respectively. The EPA (1980) performed analyses of the toxicity of 1,2- and 1,4-DCB on Fathead Minnows during the embryo-larvae stage (ELS). Results of this work indicate that this may be the most sensitive stage. Values for ELS studies range from 0.56 and 2.5 mg/L (EPA, 1980). Only a few studies have been performed to examine the toxicity of DCB on freshwater invertebrates. The 48-hour LC-50s for 1,2-DCB and 1,4-DCB were 2.44 and 11.0 mg/L, respectively, in Daphnia magna (EPA, 1978). The Midge was less responsive to these pollutants with a 48-hour LC-50 of 11.76 and 13.0 mg/L for 1,2-DCB and 1,4-DCB, respectively (EPA, 1978). Studies in algae show that reduction in chlorophyll a and reduction in cell number occur at roughly 90 to 100 mg/L (EPA, 1978).

Very little data exist on the toxicity of DCBs to saltwater organisms. From the existing data, it appears that shrimp are more sensitive than fish and plant species. Two species of saltwater fish have been tested: the Tidewater Silverside and the Sheepshead Minnow. The 96-hour LC-50 for 1,2-DCB was 7.3 mg/L for the Tidewater Silverside. The 96-hour LC-50's for 1,2-DCB and 1,4-DCB in the Sheepshead Minnow are 7.66 and 7.44 mg/L, respectively. Thus, it appears that saltwater fishes are less sensitive to the DCBs than freshwater fishes.

In Mysid Shrimp, the 96-hour LC-50s for 1,2-DCB and 1,4-DCB were 1.97 and 1.94 mg/L, respectively.

In algae, 96-hour EC50's (measurements of 50 percent reduction in chlorophyll and cell number) range between 44 and 59 mg/L.

Bioconcentration of DCBs may, however, cause chronic toxic reactions. In Bluegills, the steady-state bioconcentration factors (BCF) for the 1,2-DCB, 1,3-DCB and 1,4-DCB are 89, 66 and

60 , respectively (EPA, 1978). Neely, et al. (1974) calculated the steady-state BCF in Rainbow Trout; his calculations show a BCF of 210.

In summary, reported wastewater effluent DCB levels are so small relative to reported DCB toxicity levels that instream toxic effects from DCB in effluents are unlikely to occur.

Effects of DCB's on Humans.--A number of studies report the presence of DCBs in human tissues. For instance, West and Ware (1977) detected DCBs in human blood in residents of New Orleans. Morita, et al. (1975) report the presence of 1,4-DCB in human adipose tissue.

Most of the clinical cases of DCB poisoning reported have been through inhalation (16 of 22) (Girard, et al., 1969; Sumers, et al., 1952; Weller and Crellin, 1953; Perrin, 1941; Cotter, 1953; Gadrat, et al., 1962; Petit and Champeir, 1948; Nalbandian and Pierce, 1965; Campbell and Davidson, 1970; Downing, 1939; Frank and Cohen, 1961; Ware and West, 1977). The quantitative efficiency of absorption via inhalation has not been determined.

Three of the above 22 cases of poisoning resulted from ingestion but no quantitative absorption efficiency has been determined via this route (Campbell and Davidson, 1970; Frank and Cohen, 1961; Hallowell, 1959). Animal experiments indicate that GI absorption of DCBs is rapid since effects, excretion and metabolites have been observed within 1 day of oral exposure (Rimington and Ziegler, 1963; Azouz, et al., 1953; Poland, et al., 1971).

Human and animal studies have indicated absorption via dermal exposure. Three of the 22 clinical cases involved dermal exposure (Girard, et al., 1969; Dowing, 1939; Nalbandian and Pierce, 1965). The dermal application of 1,2-DCB (5 times twice daily applications) to the abdominal skin of rats caused dermal absorption (West and Ware, 1977).

Because of low water solubility and high lipid solubility, DCB's should be able to cross barrier membranes (West and Ware, 1977). This would allow the DCB's to be widely distributed in various tissues. Lipid soluble halobenzenes may accumulate in the body and reach toxic levels and also may recirculate for long periods (West and Ware, 1977). The clinical and experimental data indicate wide distribution resulting in changes in blood and blood chemistry, neuromuscular function, and liver and kidney structure and function.

Numerous studies of acute and chronic toxicity of DCBs have been reported in humans as well as laboratory animals. Because of their wide circulation in the body, DCBs affect numerous processes. They do not appear to be site specific like other chemicals discussed in this report (e.g., chlorophenols). All of the available literature are either follow-up studies of human ingestion or inhalation studies; and, in all cases, exposure levels do not correspond to the low level, chronic exposure found in drinking water.

Recent work using mixed isomers and 1,2-DCB failed to demonstrate carcinogenic response (EPA, 1980; Varshavskaya, 1967b). Earlier work, however, seems to indicate that 1,4-DCB may increase the rate of leukemia in rats and may promote cancer in irradiated mice (Murphy and Sturm, 1943; Parsons, 1942).

Anderson, et al. (1972) reported that 1,2-DCB was not mutagenic in the Ames test. However, several authors have reported chromosomal breakage and cell abnormalities in plants exposed to DCBs (Carey and McDonough, 1943; Sharma and Bhattacharyya, 1956; Sharma and Sarkar ,1957).

5-Chlorouracil--

Among the volatile chloroorganics found in the chlorinated effluents of wastewater treatment facilities is 5-Chlorouracil (5-CU) (Jolly, 1973, 1974, 1975; Jolley, et al., 1976; Glaze and Henderson, 1975). Because of the possible incorporation of 5-CU into nucleic acids and the potential mutagenicity and/or carcinogencity, this compound was selected for review of its known toxicity.

Effects on Aquatic Organisms.--There are only three available reports on the toxicity of 5-CU on carp eggs and embryos. There is some disagreement in the results of these studies which renders their interpretation difficult. Eyman, et al. (1975) studied the effects of 5-CU on embryos one hour after hatching and demonstrated increased mortality (1.2 to 4.3%) at concentrations ranging from 0.5 to 10.0 mg/L. Gehr, et al. (1974) demonstrated reduced hatching success in carp eggs exposed to 7.0 mg/L 5-CU. In contrast, Trubalka and Burch (1978 and 1979) studied carp embryos and were unable to observe any effects of 4-7 day exposures to 5-CU concentrations ranging from 0.01 to 100 mg/L.

Two studies on the effects of 5-CU in *Daphnia magna* have appeared in the published literature. Gehrs, et al. (1972) reported delayed and reduced production of offspring in *Daphnia* exposed for 7 days to 0.01 mg/L 5-CU; while Gehrs and Southworth (1976) reported no change in median survival time in *Daphnia* exposed to 5-CU in concentrations ranging from 0.01 to 100 mg/L. Clearly, one can tentatively conclude that in *Daphnia* reproduction is impaired at levels considerably below mortality; thus, reproductive impairment could be the factor which limits or decreases population size if concentrations reach critical levels.

Johnson, et al. (1977) carried out extensive studies on the incidence of mortality in a marine species, the Spotted Sea Trout. They studied eggs and larvae exposed to a variety of concentrations. The results of their work are shown in Table 14. These results show that 2-hour eggs are generally more sensitive than 10-hour eggs. Larvae are less sensitive than 2-hour eggs at 10 and 100 mg/L; at all other concentrations larvae display either similar or slightly higher mortality than 2-hour eggs.

TABLE 14. EFFECTS OF 5-CHLOROURACIL
ON SPOTTED TROUT EGGS AND LARVAE

Concentration mg/L	Percent Mortality		
	2 hr. eggs	10 hr. eggs	1 hr. larvae
100	100.0	44.5	56.5
10	59.0	24.5	30.0
5	7.5	12.0	10.0
1	5.5	11.5	9.0
0.5	5.0	17.5	6.4
0.1	2.0	4.0	2.0
0.01	0.5	3.0	0.5

Note - exposure temperature = 25^{o} C
test type, static, concentrations not measured
test duration = 48 hours

Gehrs and Southworth (1976) studied the toxicity of 5-CU, 4-chlororesorcinal and a complex mixture of chloroorganics. Their results show possible antagonistic effects on Daphnia.

Effects of 5-CU on Humans.--5-CU was found to be mutagenic in *E. coli* WP-1; however, 1 gram/liter of 5-CU in the drinking water of mice for more than a year produced no observable genetic or somatic effects (Cumming, 1976). This concentration approaches the maximum water solubility and is about 1,000,000 times greater than the estimated environmental level. Cumming (1976) reported that 5-CU was incorporated into the DNA in the livers and testes of mice but did not examine these or other sites for genetic damage. Cumming (1976) tested for dominant lethal mutations in male mice mated with unexposed females, but this test showed no significant results. Cumming (1976) used the specific locus test to measure recessive visible mutations at seven loci in mice; in this study, no mutations were observed.

The carcinogenicity and teratogenicity of 5-CU are not known.

Summary of Chlorination Byproduct Toxicity.

Table 15 summarizes reported wastewater effluent levels and measured effects ranges for each of the chlorination products discussed above. Several important conclusions can be drawn from these data.

1. For all byproducts except TRC, effluent levels are below levels known to be acutely toxic. Thus, any stream dilution would reduce concentrations below those reported to have acute toxic effects.

2. Effluent levels of TRC are within the concentrations known to have acute toxic effects. Thus, even with stream dilution TRC will be expected to have acute toxic effects.

TABLE 15. EFFLUENT LEVELS AND EFFECTS RANGES FOR SELECTED WASTEWATER CHLORINATION BYPRODUCTS

Byproduct	Reported Effluent Levels (mg/L)	Measured Effects Range (mg/L)
Total Residual Chlorine	1-8	0.001-10
Chloroform	0.012-0.020	1.0-300
Tetrachloroethylene	0.004	10-800
Trichloroethylene	0.01-0.04	1.0-80
Chlorophenols	0.0005-0.03	0.01-500
Dichlorobenzenes	0.01	1.0-200
5-chlorouracil	0.004	0.01-100

Note - Upper limits of ranges indicate upper limits of testing.

Please note, the effects ranges in Table 15 include only acute toxic effects - mostly LC-50 data. These data do not include long-term chronic effects in aquatic organisms. Because of bioaccumulation, seemingly low levels of a chemical in effluents and receiving stream water may have biological significance.

CHLORINATION/DECHLORINATION

INTRODUCTION.

An alternative to releasing a chlorinated wastewater effluent with a high residual chlorine (TRC) level which may be toxic to aquatic life is to dechlorinate or to remove the residual chlorine. The process of chlorination/dechlorination thus involves chlorination to disinfect the secondary wastewater followed by a dechlorination step to remove or reduce the residual chlorine remaining from the disinfection step. Although dechlorination has been used successfully in a variety of drinking water situations, it has only recently been investigated for wastewater.

There are several common methods of dechlorination: physical adsorption using activated carbon; chemical reduction using sulfur dioxide (SO_2), sodium sulfite (Na_2SO_3), or other sulfur containing reducing agent; photochemical decomposition using ultraviolet radiation, (Seegert, 1978) and holding ponds for the dissipation of chlorine residuals (Gan, et al., 1979). Of these methods, the use of sulfur dioxide is the most cost effective and appears to have the greatest promise for wastewater applications (White, 1972) (Gan, et al., 1979). Reasons for sulfur dioxide popularity for dechlorination are centered around its similarity to chlorine -- commercial availability as a compressed liquid, transportation modes, handling of liquified gas, and safety precautions -- as well as cost effectiveness. Due to similar physical and chemical properties for chlorine and sulfur dioxide, equipment is often interchangeable. Therefore, this risk analysis concentrated on sulfur dioxide as a dechlorinating agent.

HAZARD IDENTIFICATION.

The identification of hazards, or risks, associated with the chlorination/dechlorination wastewater disinfection process should include the risks within the production, transportation and handling, and use categories for both chlorine and sulfur dioxide. In addition, there may be identifiable risks from the combined use of the two chemical agents which may be absent when each is analyzed separately. The risks associated with the use of chlorine are treated above. Production risks were omitted from this study in the scope of work.

Sulfur dioxide is used in numerous industrial applications -- as a refrigerant, a bleaching agent, a disinfectant, a liquid solvent, and as a raw material for the production of sulfuric acid (Shreve, 1967). Thus, its present and potential use as a dechlorinating agent represents a small fraction of the sulfur dioxide produced in the United States. Neglecting the risk associated with the production of sulfur dioxide for the purpose of dechlorination will have a negligible effect on the overall risk associated with the production of sulfur dioxide.

The risks associated with sulfur dioxide will be similar to those for chlorine due to similar chemical and physical properties, and transportation and handling methods.

The primary risks associated with the transportation and handling of sulfur dioxide are:

1. human exposure to liquid sulfur dioxide;
2. human exposure, both occupational and public, to gaseous sulfur dioxide; and
3. vegetation exposure to either liquid or gaseous sulfur dioxide.

Human exposure to liquid sulfur dioxide can cause serious skin or eye burns; however sulfur dioxide vaporizes at -10°C at atmospheric pressure (Handbook of Chemistry and Physics, 1969). Thus, most exposures will be to gaseous rather than liquid sulfur dioxide.

Sulfur dioxide is a colorless gas with a suffocating odor. The principal human exposure routes are inhalation followed by eye and skin contact. The taste threshold is approximately 0.3 to 1 ppm, and the odor threshold is approximately 0.5 to 1 ppm (U.S. Department of Health, Education, and Welfare, 1970, and Faith, 1972). Sensitive individuals notice sulfur dioxide in the 1 to 2 ppm range while most people show respiratory irritation when chronically exposed to 5 ppm concentrations. Severe bronchospasms can be initiated in the 5 to 10 ppm range (Faith, 1972). When sulfur dioxide contacts atmospheric moisture, sulfurous acid (H_2SO_3) may be formed which is a toxic irritant and is also highly corrosive.

Different plant species vary considerably in their susceptibility to exposure to gaseous sulfur dioxide. The response to sulfur dioxide is also dictated by factors such as temperature, humidity, plant age, and soil moisture. Acute exposures to sulfur dioxide cause leaf discoloration and destruction. Threshold concentrations start at about 1.0 ppm for a one hour exposure (alfalfa). Chronic exposures result in yellowing of leaf tissue, leaf drop, lesions, and suppression of growth. Chronic symptoms occur at much lower concentrations, typically less than 0.03 ppm. Low concentrations of sulfur dioxide can also react synergistically with ozone to cause injury to some sensitive plant species (Faith, 1972).

The primary risks associated with the use of chlorine/sulfur dioxide as a mechanism of disinfection and dechlorination are:

1. human and aquatic life exposure to residual chlorine,
2. human and aquatic life exposure to chlorine reaction products,
3. human and aquatic life exposure to sulfur dioxide reaction products,
4. human and aquatic life exposure to both sulfur dioxide and chlorine reaction products,
5. aquatic exposure to low dissolved oxygen effluent, and
6. aquatic exposure to low pH.

The risks associated with residual chlorine and chlorine reaction products are discussed above.

When sulfur dioxide is dissolved in water, sulfurous acid (H_2SO_3) is formed, which is the actual dechlorinating agent. Sulfurous acid reacts virtually instantaneously with both free and combined chlorine to form chlorides, sulfates, and bisulfates as the primary reaction products (White, 1972). Sulfurous acid is also a moderately strong acid which will dissociate to form bisulfite (HSO_3^-) and sulfite (SO_3^{2-}). Research is required to identify the list of inorganic and organic reation products associated with the use of sulfur dioxide as a dechlorination agent. Research is also required to determine what effect, if any, sulfur dioxide has on the chlorine reaction products formed in the chlorination step or those which previously existed in the wastewater effluent prior to chlorination. Nevertheless, the primary products from dechlorination should be inorganic salts such as chlorides, sulfates, and sulfites which have been shown to be nontoxic to aquatic life at concentration levels usually associated with wastewater treatment (Gan, 1979).

The use of a chemical dechlorination agent like sulfur dioxide requires an accurate and fail-safe injection and monitoring system (Seegert, 1978). The addition of too little sulfur dioxide can lead to a chlorine residual remaining in the wastewater effluent which can be toxic to aquatic life at very low levels. The monitoring of the residual chlorine before dechlorination is the usual method of varying the required sulfur dioxide (called feed forward control). For feedback control, the limit of detection for residual chlorine analysis may be above the maximum recommended residual chlorine level required for the protection of aquatic organisms. The addition of too much sulfur dioxide can result in injury to aquatic life. To prevent low dissolved oxygen, an aeration process may be required downstream from the dechlorination step (U.S. Environmental Protection Agency, 1976). However, Gan et al.(1979) found that reaeration is not necessary, because there was negligible reduction in dissolved oxygen as a result of sulfur dioxide dechlorination.

SECTION 5

HAZARD INDENTIFICATION FOR OZONE

Ozone may be considered one of the most promising alternatives to chlorine as a wastewater disinfectant. This section deals with the identification and quantification of the hazards associated with ozone as a wastewater disinfectant.

The identification of hazards, or risks, associated with ozone is performed by considering the production, transportation and/or handling, and use cycles in a manner similar to that for chlorine. The instability of ozone dictates that it be produced on-site; thus, primary risks associated with ozone can be limited to on-site production and use. Knowledge of treatment plant facilities for the production and use of ozone allows detailed hazard identification.

The quantification of risks associated with ozone is much more difficult than the quantification process for chlorine. Although ozone has been extensively used as a disinfectant for drinking water treatment in Europe, there is very limited use of ozone for wastewater treatment. Furthermore, in the United States chlorine is almost universally used for disinfecting water and wastewater, and ozone's limited use in the United States has been for taste, odor, and color control in the treatment of water (Layton, 1972). The use of ozone as a wastewater disinfectant is expanding in the United States (Venosa, 1972; Hais and Venosa, 1978), but experience is moderate.

The primary risks associated with the use of ozone as a wastewater disinfectant are:

1. Human and vegetation exposure to gaseous ozone;
2. Human and aquatic exposure to residual ozone;
3. Human and aquatic exposure to ozonation reaction products

ON-SITE USE HAZARDS

The primary on-site risks associated with the production of ozone are:

1. human exposure, both occupational and public, to gaseous ozone;
2. vegetation exposure to gaseous ozone in the vicinity of wastewater treatment facility; and
3. human contact with high electrical voltage.

Human exposure to gaseous ozone is associated with headaches, irritation of respiratory tract, and irritation to eyes with changes in visual acuity. For sensitive individuals, irritation from ozone starts near the odor threshold, 0.02 to 0.05 ppm by volume. Current human exposure standards for ozone are 0.1 ppm for a period not to exceed 8 hours. This requirement dictates efficient treatment facility design to collect and destroy exhaust ozone. One plant has experienced ambient concentrations of ozone in the 15 to 30 ppm range during initial start-up conditions (Rakness and Hegg, 1979). Elevated ozone concentrations can also be injurious to vegetation in the vicinity of the treatment facility.

Ozone is partially soluble in water, and it is difficult to obtain concentrations of more than a few milligrams per liter in aqueous solutions under normal conditions of pH, temperature, and pressure. Thus, at high applied dosages as might be used in the disinfection of industrial and municipal wastewater, the atmosphere above the contact tanks will be rich in both oxygen (O_2) and ozone (O_3). If the ozone concentration in the offgas is not reduced by mechanical or chemical means, the discharge offgas remains rich in ozone. Ozone is 1.5 times as dense as oxygen and has a long half-life in the ambient atmosphere of approximately 12 hours (Miller, et al., 1978). Consequently, there is a real possibility of high atmospheric concentrations of ozone in the vicinity of the wastewater treatment facility, which can be injurious to both plant and animal life.

The limited data available on the use of ozone for wastewater disinfection prevent a quantitative assessment of the accident rate for workers in wastewater treatment facilities that use ozone. Furthermore, although the Europeans have considerable experience using ozone as a disinfectant for potable water, there are no parallel data for accident statistics. In fact, ozone in Europe is not seen as a particular cause of accidents or injuries (Bres, 1981).

PROCESS REACTION PRODUCT HAZARDS

The hazards associated with ozonation reaction products must be considered due to the variety and toxicity of reaction products associated with the use of chlorine. Although the consensus in the literature (Hais and Venosa, 1978; Winklehaus, 1977) indicates that ozone produces fewer harmful intermediate and end products than does chlorine, the fact remains that very little work has been done to identify ozone reaction mechanisms and to identify ozone reaction products under field conditions.

Ozone can react by several mechanisms in wastewater depending on pH, presence of metals, and types of organic compounds present (Winklehaus, 1977). In general, oxidation reactions of organic compounds with ozone are not complete (formation of H_2O and CO_2) thus leading to the production of both stable and unstable intermediate and end product compounds. Smaller, saturated organics are less reactive (more refractory) to ozonation. Ozone products identified under field and laboratory conditions include low molecular weight alkanes, aldehydes, organic acids, and heterocyclics (Chappell, et al., unpublished; Kuo, et al., 1977). Although ozonation end products may accumulate after repeated cycles of water reuse and thus constitute a risk, there is little known about ozonation products and their toxicity.

Effects of Ozone Residual on Aquatic Organisms

Hubbs (1930) investigated several water purification processes for treating water supplies for fish. One of the processes studied was ozonation. The responses of several fish species included altered locomotive and respiratory movements, followed by loss of equilibrium and wild swimming with alternating quiescent periods when the fish rested on its side or back. The quiescent periods lengthened and finally terminated in death. Fish could recover from the symptoms of altered locomotive and respiratory movements but not from lost equilibrium even when they were transferred to a suitable water supply. Residual ozone concentrations as low as 0.09 mg/L were lethal to fish in flow-through assays. Crayfish died after exposure to 1.16 mg/L residual ozone and unspecified planktonic and bottom invertebrates were killed by residual ozone concentrations of 1.25 mg/L. Oxygen super-saturation, pH, and CO_2 content were eliminated as possible causes of mortality. The methods used by Hubbs to measure the ozone residuals (unbuffered potassium iodide) may have led to errors in reported levels (Schechter, 1973).

Giese and Christensen (1954) studied the effects of residual ozone in static tests on freshwater protozoa and rotifers. The organisms were studied in minute volumes of water under a microscope, and measurements of ozone residuals were not done. The residuals were probably low but the concentrations were high enough to kill the organisms.

Giese and Christensen (1954) studied sea urchin (Strongylocentratus purparatus) and marine worm (Urechis caupo) eggs in unmeasured ozone concentrations and found changes in the membranes and cortex of the animals.

McLean, et al. (1973b) studied the effects of ozonated seawater on the eggs of the commercial American oyster (Crassostrea virginica). The residual ozone concentration was estimated to be less than 0.20 mg/L in a static bioassay. Eggs spawned in the ozonated seawater exhibited an increase in fertilization defects (decreased polyspermy and parthenogenesis) and intercellular abnormalities (retarded meiosis and cleavage, irregular polar bodies and abnormal nuclear cleavage) compared to eggs spawned in unozonated seawater.

A residual ozone concentration of 0.01 to 0.06 mg/L was demonstrated to cause 100% mortality within four hours to Rainbow Trout. But when the same lakewater for hatchery use was aerated for 11 minutes prior to delivery to the trout tanks the mortality was eliminated (Rosenlund, 1975).

Barnacles (Balanus sp.) which were continuously exposed to ozonated seawater (with a residual of 0.4 to 1.0 mg/L) died after several days (up to a week) exposure. (Mangum and McIlhenny, 1975)

Exposure to a residual ozone concentration of 0.1 mg/L for 5 minutes was lethal to marine phytoplankton (Skeletonema lostatum, Chlorella sp., Nannochloris sp., and Monochrysis lutheri) within 24 hours. Crab zoea exposed for one minute to 0.08 mg/L of residual ozone showed a 0-20 percent mortality after 24 hours and 30-40 percent mortality after 48 hours. A one minute exposure to 0.2 mg/L residual ozone resulted in 100 percent mortality within 24 hours for crab megalops. Atlantic Silverside (Menidia menidia) exposed to 0.08-0.2 mg/L residual ozone were killed within 30 minutes (Toner and Brooks, 1975).

Acute and chronic flow-through laboratory bioassays were conducted with domestic secondary wastewater effluent disinfected by ozonation and diluted by lake water. Seven fish species (Brook trout, Coho salmon, Fathead minnow (fish and eggs), Whitesucker, Walleye, Yellow perch, Largemouth bass) and six invertebrates (Amphipods, Stonefly, Caddisfly, Crayfish, Operculate snail, and Pulmonate snail) were tested for seven days in acute bioassays. Two generations of one species of fish (Fathead minnow) and two invertebrate species (Daphnia magna, and the amphipod, Gammarus pseudolimnaeus) were assayed in a chronic test. A residual ozone concentration of 1-2 mg/L disappeared so rapidly from the bottom of the ozone contact column to the test-tanks that no measureable levels were detected in the test water for both tests. No measureable toxicity to aquatic life was found from either long- or short-term exposures. When procedures were adjusted to shorten the retention time so that a residual ozone concentration could be maintained continuously in the effluent, an acute exposure to 0.2-0.3 mg/L was lethal to Fathead minnows after 1-3 hours (Arthur, et al., 1975).

Increased hatchability and improved survival was reported for fry from Rainbow trout eggs incubated in recirculating aquarium water treated with ozone and deozonated with activated carbon to a residual below 0.10 mg/L (Benoit and Matlin, 1966).

Rosenkranz, et al. (1978) reported that the 96-hour LC50 for adult White perch (Morone americana) exposed to ozone-produced oxidants (OPO) was 0.22 mg/L. A significant decrease in blood pH and an increase in hematocrit over the test period (96 hours) were observed in fish exposed to 0.10-0.15 mg OPO/L. Histological changes in the gills of fish exposed to 0.01-0.15 mg OPO/L over 24 hours were seen, but gill repair was evident after a 14 day recovery period.

Richardson, et al. (1978) exposed 12-hour old Striped bass (Morone saxatilis) eggs to a range of 0.005-0.10 mg OPO/L, then evaluated the effects on egg development at 24 and 42 hours after fertilization and the effects on two prolarval stages (24 and 48-hour post hatch). The results indicated that OPO were more toxic in freshwater after exposures in both fresh and estuarian water. The eggs were more resistant than the prolarvae. Delayed hatching occurred in eggs exposed to 0.05 and 0.10 mg/L OPO (estuarine water), and the delayed hatch organisms survived better than the hatched larvae at the same stage of development. Wedemayer, et al. (1979) found that the acute toxicity curve for dissolved ozone yielded a 96-hour LC50 of 0.093 mg/L O_3 for juvenile Rainbow trout. The authors reported death due to acute exposure was most likely due to severe gill lamellar epithelial tissue destruction accompanied by massive hydro-mineral imbalances. Chronic tests showed little damage at 0.002 mg/L and some histopathological changes in gill tissue at 5 ug/L O_3.

The 24-hour LC50 for bluegill (Leponis macrochirus) was 0.06 mg/L and periodic dosing of six 30-minute exposures, 8 hours apart, gave an LC50 of 0.32 mg/L. Bluegills held in concentrations between 0.01 and 0.02 mg/L showed irregular respiration, decreased activity, cessation of feeding, and random mortality during a 6-week exposure (Paller and Heidinger, 1979).

Ward, et al. (1976) conducted bioassays using ozonated wastewater obtained from a Michigan treatment plant that receives mostly domestic wastes. Long-term life cycle toxicity tests were conducted with several cold and warm water fish species and one invertebrate species. Fathead minnows were exposed to filtered ozonated wastewater diluted from zero to 100% with wellwater and with an O_3 residual from 0.00 to 0.016 mg/L O_3 for 30 days (first generation) and 60 days (second generation). The first generation of fish exhibited no lethal effects attributable to residual ozone. The second generation of fish reared in about the same residual ozone concentration (0-0.013 mg/L) and dilutions as the first generation exhibited no definite lethal response. The authors suggest that long-term exposure to ozonated effluent would not be toxic or lethal to Fathead minnows. In the same study no significant differences in length or weight were observed over the life cycles of two generations of Fathead minnows. The same species of fish reared in various concentrations (0-0.016 mg/L) and dilutions (0-100%) of ozonated effluent showed no adverse reproductive effects. There appeared to be no adverse effects on the hatchability of eggs produced and incubated in water with a ozone residual of 0.00 to 0.01 mg/L.

In experiments where 14 species of cold and warm water fish were exposed to 100% ozonated effluent within 10 minutes after disinfection, goldfish and fathead minnows survived an ozone residual of 0.047 to 0.185 mg/L for 7-15 days. Under similar conditions lake trout fingerlings suffered 100% mortality after five hours at an ozone residual of 0.322 mg/L. The other species tested showed no mortality to ozone concentrations of 0.002 to 0.38 mg/L but due to ozone generation problems some of these species were not tested for 96-hours.

Ward, et al. (1976) found Daphnia magna (less than 24 hours old) had a 30% mortality when exposed to 100% ozonated effluent (residual O_3 0.03 mg/L) for 96 hours.

Ward, et al. (1977) used the same methods as in their previous study (Ward, et al., 1976) to examine the acute and chronic toxic effects on several freshwater fish and invertebrates. The effluent to be ozonated was from a secondary treatment plant in Wyoming, Michigan that receives 35-45% of its waste from light industry (metal plating plants, dairy products) and 55-65% from domestic sources.

The results of the life cycle test were much the same as in the previous study. Adult fathead minnows exposed to residual ozone concentrations at 0.003 to 0.01 mg/L showed no mortality due to the ozone. Fathead minnow fry exposed to 0.001 mg/L O_3 over 60 days produced poor survival data that was inconclusive due to poor water quality not associated with the ozone disinfection.

Ward (1976) reported that residual ozone in ozonated secondary effluent diminishes to a concentration less than 0.01 mg/L within about 15 minutes after dosing. Arthur, et al. (1975) also found in their toxicity studies with the fathead minnow that an ozone dose rate of about 5.7 +- 1 mg/L was needed to disinfect and attain a residual of 1 mg/L . At an ozone residual level of 10 mg/L dissipation of ozone in the effluent was so rapid that levels in the test tanks were unmeasureable and non-toxic. Rosenlund (1975) also reported that water with a residual of 0.01 to 0.06 mg/L became nontoxic when aerated for 11 minutes before introduction to fish.

Bioassays to determine ozone levels and doses to prevent unwanted introduction of fishes into a reservoir were conducted by Coler and Asbury (1980a). Larvae and, in some species, eggs were exposed to ozonated lake water in static and continuous tests. The 24-hour LC50 values ranged from 4.0 mg/L for channel catfish eggs to 0.19-0.31 mg/L for rainbow trout larvae. In another study by the same authors (1980) larvae and, in some fish, eggs were assayed in a flow-through system at different time periods and different concentrations to determine LC50 values for residual ozone exposures. In both of the above studies eggs were more tolerant than larvae to ozone concentrations.

Summary of the Effects of Residual Ozone

Residual ozone is toxic to aquatic organisms even at low concentrations. The general pattern of toxicity for freshwater fish appears to begin at a concentration of about 0.001 mg/L with gill damage and loss of equilibrium. These conditions may result in the subsequent death of some fish but unless the concentration reaches the 0.01 mg/L range, where 50% to 100% mortality may occur, most fish should recover with the dissipation of the ozone.

The larvae of most species of fish seem to be more sensitive to ozone than fish eggs. Adverse effects appear in the 0.1 mg/L range for larvae and 1 mg/L range for eggs. The tolerance to ozone by fish eggs appears to be associated with the membrane. Benoit and Matlin (1966) indicated that ozone may act on the membrane only resulting in death if the membrane or gelatinous matrix is disrupted causing lysis or leakage. Since adult or fry fish will experience adverse effects at a concentration at least one order of magnitude lower than the effect levels for eggs and larvae, the early life stages of fish should be protected if residual ozone levels are maintained below 0.001 mg/L range.

Freshwater invertebrates seem to be less sensitive to residual ozone than fish. Mortality for most species does not begin until the concentration reaches 0.01 mg/L range.

Laboratory estimates of ozone decay rates in Connecticut River water at 9 and 22 degrees Celsius showed a linear logarithmic decrease from 2.0 to 0.05 mg/L in 30 and 60 minutes, respectively (Coler and Asbury, 1980). Thus, when ozonated effluents are aerated and then diluted rapidly, concentrations of ozone will dissipate below the toxic levels.

Some studies indicated that ozonated water may be beneficial to the growth and survival of fish eggs and fry after the residual has been removed or dissipated (Benoit and Matlin, 1966, Ward, et al., 1976). The benefit may be due to the increase in the dissolved oxygen (DO) content of ozonated water. Supersaturation of oxygen in water does not seem to have great toxic effects on fish at long exposure times (Wolke, et al., 1974; Boack, et al., 1976; Nebeker, et al., 1979).

Although the data indicate that residual ozone is toxic to aquatic life at low concentrations, it is not very stable in water and will likely dissipate to concentrations below the toxic levels before any adverse effects on aquatic life are noted. Ozone residuals are typically measured at the exit side of the contact basin which may be some distance upstream from where the disinfected wastewater effluent is discharged into the receiving body of water. In addition, the residence time for dissolved ozone in aqueous solutions is very short, since even in a nonreactive environment of distilled water, the half-life of ozone is only 20 to 30 minutes (Miller, 1978).

Ozone remaining in effluents of ozone treatment plants will dissipate long before any such water would be incorporated into the drinking water supply system. Thus, the human health risk from ozone residuals in wastewater effluents is insignificant.

Effects of Ozone By-products on Aquatic Organisms

The by-products of wastewater ozonation are dependent on the organics (the precursors) present in the wastewater prior to ozonation, and the number of potential precursors is very large. The ozonated effluent of the Upper Thompson Sanitation District treatment plant in Estes Park, Colorado, was analyzed for organic residuals by Chappell, et. al. (1980); and a large number of simple aliphatic and aromatic compounds were found. In order to develop a sense for the hazards associated with the ozonation by-products, the organic compounds discussed below were selected for research in the literature.

n-heptane.--Several studies were cited in Vershlueran (1977) regarding n-heptane toxicity. In Mosquito Fish a concentration of 5,600 mg/L had no apparent effect whereas toxicity was reported at 1,000 mg/L. The 24-hour LC-50 in Mosquito Fish was reported to be 4,900 mg/L. In Goldfish the 24-hour LD-50 was reported at 4 mg/L. In young Coho Salmon no significant mortality was induced at concentrations below 100 mg/L after 96 hours in artificial seawater at 8 C. These data obviously demonstrate a wide range of toxicity.

n-octane.--Verschlueran (1977) reported only one study of toxicity for n-octane. In this study young Coho Salmon exhibited no mortality in concentrations of less than 100 mg/L when exposed for 96 hours.

n-hexanal.--No data available on aquatic toxicity.

m-xylene.--Verschlueran (1977) found the 24-hour LC-50 for Goldfish to be 16 mg/L. Hann and Jensen (1974) reported that the LC-50 was 10 to 100 mg/L. A few studies on the toxicity of o-xylene were available. In Goldfish, the 24-hour LC-50 for o-xylene is 13 mg/L (Birge, 1979). Brenneman, et al. (1976) report the 96-hour LC50 for o-xylene in Goldfish at 14 mg/L. Hann and Jensen (1974) reported that the 96-hour LC50 for Fathead Minnows was 42 mg/L. In young Coho Salmon, o-xylene produced no significant mortality in fish exposed 24 to 96 hours to 10 ppm; the 24-hour LC-50 was determined to be 100 mg/L. The alga, Chlorella vulgaris, exposed for 1 day to 55 mg/L, suffered a 50 percent decline in cell number. Walsh, et al. (1977) reported a 96-hour LC-50 for xylene (no isomer given) in Rainbow Trout at 13.5 mg/L.

n-heptanal.--No toxicity data available.

n-nonanal.--No toxicity data available.

Ozone by-products would be produced in such small quantities (ppt) (Chappell, et. al., 1980) in wastewater plant effluents that the human exposure risks are negligible. Furthermore, dilution of these products by receiving streams results in even less risk. Finally, the toxic dose-response levels for ozone by-products are orders of magnitudes greater than the expected effluent levels so adverse effects to aquatic organisms are not likely to occur.

SECTION 6

HAZARD IDENTIFICATION FOR ULTRAVIOLET RADIATION

The use of ultraviolet radiation to disinfect wastewater is now receiving attention as a possible alternative to the use of chlorine. To become a viable alternative to chlorine in the disinfection of wastewater, ultraviolet radiation must be safe and effective.

The identification of hazards, or risks, associated with ultraviolet radiation is performed by considering the production, transportation and/or handling, and use cycles. Ultraviolet radiation is produced on-site, which eliminates the transportation and handling problems. Risks associated with the use of ultraviolet radiation as a wastewater disinfectant are human and aquatic exposure to insufficiently disinfected wastewater effluent and human and aquatic exposure to ultraviolet radiation reaction products. Primary risks associated with ultraviolet radiation are limited to on-site production and use.

The quantification of risks associated with the production and use of ultraviolet radiation is very difficult when only considering published or historical information. Although there has been some effort in using ultraviolet radiation for the sterilization of potable water and the bactericidal effects of ultraviolet radiation have been known for many years, there has been very little application of ultraviolet disinfection of secondary wastewater on any scale. Most of the potable water sterilization systems using ultraviolet radiation have been small in scale and drawing a parallel comparison between wastewater and potable water again will be difficult.

ON-SITE USE HAZARDS

The primary on-site risks associated with the production of ultraviolet radiation are:

1. human exposure to ultraviolet radiation
2. human exposure to ozone

3. human contact with high electrical voltages.

Although direct exposure to ultraviolet radiation is improbable in a well designed wastewater treatment facility, accidental exposures to treatment plant personnel would carry significant risk to human health. Human exposure to ultraviolet radiation primarily affects the skin and eyes. Reddening, or burning, of the skin due to ultraviolet radiation exposure is most pronounced at a wavelength of 260 nm which is nearly identical to the major radiation wavelength, 254 nm, produced by the mercury discharge lamps used in disinfection. The severity of skin burning is a function of the total ultraviolet dosage and ranges from simple reddening to blistering and peeling of skin with possible severe secondary effects. Data indicate that ultraviolet radiation may produce or initiate carcinogenesis in human skin. Ultraviolet exposure to eyes can result in damaged corneas, impairment of visual acuity, and eye fatigue. With the exception of cornea damage, most effects are temporary (HEW, 1973).

Injurious threshold levels for ultraviolet radiation depend on the particular wavelength. To prevent reddening of unprotected skin the American Medical Association has published an exposure limit of 0.5 uWatt/square centimeter for exposure at 254 nm for up to seven hours. The American Conference of Governmental Industrial Hygenists has proposed a limit of 1,000 uWatt/square centimeter for 300 to 400 nm radiation for a period of 16 minutes to protect both skin and eyes. Since average ultraviolet disinfection dosages for wastewater are in the range of 10,000 to 100,000 uWatt-sec/square centimeter, accidental exposure to ultraviolet radiation poses a human health risk.

Ultraviolet radiation is capable of producing ozone when oxygen, or air, is irradiated with low wavelength (<200 nm) ultraviolet radiation. When the UV lamps and quartz sleeves are maintained at the optimum temperature, air can be circulated around the lamps, and thus the exhaust air would be rich in ozone (Sheible, 1979). Waste treatment plant personnel could therefore be exposed to ozone if the exhaust air is not properly treated or vented. Ozone production from ultraviolet exposure of dissolved oxygen in the wastewater appears to be insignificant. This is primarily due to the low level of dissolved oxygen in wastewater and the absorbing nature of the wastewater thereby diminishing the ultraviolet radiation levels after a short distance from the lamp source.

RADIATION AND REACTION PRODUCT HAZARDS

Ultraviolet radiation is classified as a physical disinfecting agent. The primary germicidal effects of ultraviolet radiation are due to the absorption of UV radiation by the genetic material (deoxyribonucleic acid (DNA) and ribonucleic acid (RNA)) of the microorganisms. The resulting dimerization of pyrimidine bases in the genetic material distorts the molecule and prevents proper replication of cell material. This results in death, mutation of offspring, and inviable offspring (Sheible and Bassell, 1979). Photoreactivation is defined as restoration of ultraviolet lesions in a biological system with light of wavelength longer than that of the damaging radiation (Johnson, et al.; 1979). Thus, when ultraviolet irradiated wastewater effluent is exposed to radiation in the visible region, a repair mechanism may be activated which results in recovery of some microorganisms, and this photoreactivation represents a significant risk to human and aquatic exposure if the reactivated organisms are pathogenic.

As with all disinfecting agents, there is a possibility of producing additional chemical compounds when ultraviolet radiation is used to disinfect wastewater. The absorption of ultraviolet radiation, especially by organic compounds, can lead to free radical formation with resulting molecular rearrangements and to possible mutagenesis of the microorganisms. Only preliminary work has been done on the irradiation effects on organic compounds; however, these initial findings indicate that there is little chemical effect on the UV-absorbing constituents in wastewater (Jolley, et al.; 1979). This is a marked contrast to chlorine and ozone, both of which produce a large number of new organic compounds in the disinfection process (Jolley, et al.; 1979).

The use of ultraviolet radiation does not produce a residual. Consequently there is no risk to human and aquatic life from a disinfectant residual.

SECTION 6

NO DISINFECTION

This section investigates the alternative of discharging wastewater that has not been disinfected. The analysis is focused on the hazards created by discharging pathogenic organisms and the associated hazards posed to humans since that is the primary reason for adopting wastewater disinfection practices. This focus on the human risks was reinforced when no discussion of hazards to the aquatic environment from pathogenic organisms was found in the literature.

HAZARD IDENTIFICATION

Pathogenic organisms, by definition, cause disease in human beings. Waterborne transmission of these disease-causing organisms can occur via four pathways:

1. direct ingestion of untreated water,
2. direct ingestion of treated drinking water,
3. ingestion of aquatic food species infected with pathogens absorbed from contaminated waters, and
4. invasion resulting from skin contact with contaminated water.

The first three pathways are sometimes classified as the fecal-oral route. The second pathway described above occurs when a drinking water treatment system fails or the integrity of the water distribution system is violated. The fourth pathway is likely to result in skin, mucous membrane, or urinary tract infections but is seldom implicated in gastrointestinal illness in the United States. Since most published research focuses on the three fecal-oral pathways the health effects of this fourth pathway is not well documented. The risk of disease by exposure to wastewater effluent in recreational water, especially non-disinfected effluent, is not well established on epidemiological grounds; however, recent work by Cabelli(1981) has demonstrated a cause-effect relationship via this pathway.

The following discussion describes the major microbes and parasites suspected of being transferred by the fecal-oral route. Bacteria, viruses, and protista are the most common causes of waterborne disease outbreaks. Fungi are rarely implicated.

Bacteria.

Many of the bacterial diseases may be transmitted by polluted water, especially those due to *Salmonella* and *Shigella*. *Salmonella typhimurium* is the species most often implicated in waterborne disease outbreaks. Symptoms include nausea, vomiting, and fever. Death is possible without timely and adequate treatment.

Other species implicated are *Salmonella typhi* and *Salmonella paratyphi*. *Salmonella typhi* is responsible for an acute disease characterized by fever, malaise, anorexia, bradycardia (slow heart rate),and enlargement of the spleen. Complications may extend to the lymphoid tissues, intestinal hemorrhage, mental dullness, and slight deafness. The fatality rate ranges from 2-3% with antibiotic therapy. The disease is spread by food or water contaminated with feces of a carrier or patient. A carrier is a person who harbors the organism but remains asymptomatic. *Salmonella paratyphi* may present a clinical picture similar to *Salmonella typhi*. The disease is primarily transmitted by food, especially milk products or shellfish. The proportion of cases that are recognized clinically is small, that is, those that present symptoms severe enough to require medical attention.

Shigella causes a disease characterized by fever, cramps, and abdominal pain. *Shigella sonnei* is the species most commonly isolated.

Invasive strains of *enteropathogenic Eschericia coli* behave much like *Shigella*. *E. coli* is part of the normal intestinal flora. Biochemically and morphologically, the enteropathogenic strains behave the same as non-pathogenic strains. Serological identification is necessary. Newborns are the most susceptible to infection by *enteropathogenic E. coli*, having fatalities up to 40%.

Cholera is an acute intestinal disease due to the organism *Vibrio cholerae*. The disease is characterized by watery stools, vomiting, dehydration, and circulatory collapse. In untreated cases, the fatality rate may exceed 50%; in treated cases, the fatality rate is less than 10% (Okun and Ponghis, 1975). Many asymptomatic cases occur and, as mentioned previously, not all individuals exposed to disease-causing doses of the organism elicit disease symptoms. Primarily, the disease is transmitted by ingestion of water or food contaminated with feces containing the organisms.

Another Vibrio species, Vibrio parahemolyticus, causes diarrhea and abdominal cramps. Though rare, the disease is most often caused by ingestion of raw seafood.

An organism called Campylobacter fetus sub. jejuni (also called Vibrio fetus) has been found to cause gastroenteritis. Special culture media and modified techniques are necessary to identify this organism so its role in waterborne disease was not recognized until recently. It is possible that this organism was not detected in previous gastroenteritis outbreaks because the proper techniques and media were not used to isolate the organism. It appears that Campylobacter may be isolated from the feces of patients with diarrhea as frequently as Salmonella or Shigella (Sack, et al., 1980).

An organism known as Yersinia enterocolitica may cause acute gastroenteritis, bloody diarrhea, and fever. It may also cause pseudoappendicitis. The organism has been isolated from a wide variety of animals including cows, beavers, and oysters. It has also been recovered from rivers, lakes, and well water. Unlike the previously described bacteria, this organism grows very well at refrigeration temperatures.

Viruses.

The viruses most likely to be found in a wastewater discharge are the human enteric virus groups Coxsackie, Polio, Echo, Reo, Adeno, and Hepatitis A. The potential consequences of human infection by these viruses are described below. More detailed discussion of these consequences can be found in a report by Benenson (1975).

Group B Coxsackie virus types 1, 2, 3, 4, and 5 have been cultured from wastewater effluent. The Coxsackie B virus is responsible for approximately 1/3 of the cases of non-fatal aseptic (nonbacterial) meningitis. Aseptic meningitis may also be due to Echo, adenoviruses, arboviruses, polioviruses, and the Coxsackie A viruses which may be found in wastewater effluent. The group B Coxsackie viruses are implicated in pleurodynia (pain in the intercostal muscles). The disorder is characterized by sudden onset with recurring chest or abdominal pain.

The Polio virus may cause a disease ranging in severity from mild, to nonparalytic, to paralytic. Fever, headache, gastrointestinal symptoms, and vague bodily discomfort are not uncommon. Other enteroviruses, such as Coxsackie and Echo, can produce symptoms very similar to the Polio virus. In a rare instance, milk has been implicated as the mode of transmission.

The parvovirus and reovirus-like particles are believed to be largely responsible for viral gastroenteritis. This disease is characterized by nausea, vomiting, diarrhea, abdominal pain, and fever. The reovirus is frequently implicated in children's diseases.

A large number of viruses are implicated in respiratory disease. Death and long-term illness due to these agents is especially prevalent in children. Adults also have a high incidence and lose many productive hours due to these agents. Symptoms range from cold-like chills, aches, and fever to severe bronchitis and pneumonia. The following viruses have been implicated: parainfluenza type 3, adenovirus types 1,2,3,4,5,7,14,21, respiratory syncytial virus, rhinoviruses, coronaviruses, coxsackie viruses types A and B, and echoviruses. Presently, transfer is thought to occur by direct contact with articles soiled by respiratory discharges, oral contact, or droplet spread; however, waterborne transfer is possible.

The Hepatitis A virus has become a major concern in recent years. The disease has an abrupt onset characterized by fever, malaise (a general feeling of bodily discomfort), anorexia (loss of appetite), nausea, abdominal discomfort, and jaundice. The disease may follow a mild course of 1-2 weeks or last several months. Transmission is believed to occur by way of the fecal-oral route. Fecal contamination of recreational water then could result in transmission of the disease.

Protista.

The most commonly implicated protozoan in waterborne disease outbreaks is Giardia lamblia. The disease caused by this organism is called Giardiasis. It is characterized by diarrhea, cramps, fatigue, and weight loss. In severe cases, malabsorption may occur. Many individuals infected with the organism are asymptomatic. The organism exists in both a cyst and trophozoite stage. As a cyst, it is very resistant to any type of environmental assault, such as chlorine dosages commonly used in water supply disinfection. Proper filtration seems to be effective in reducing outbreaks of Giardiasis.

Another protozoan, Entamoeba histolytica, may give rise to intestinal disease. In epidemics, it is believed that the disease is transmitted mainly by water containing cysts from the feces of infected individuals. Individuals who have the cysts may be asymptomatic or have acute diarrhea with chills and fever. Complications include the development of an amoebic granuloma resulting in a tumor-like appearance in the wall of the large intestine. Abscesses of the liver, lung, or brain may also result, as well as ulceration of the skin. Like Giardia, this organism exists in both the cyst and trophozoite stages. The trophozoites, being very fragile, pose less threat of disease. Filtration is effective in removing cysts.

Naeglaria fowleri, first reported in Australia in 1965, is associated with diseases acquired from recreational waters. It exists in both the trophozoite and cyst stages, and its growth may be enhanced by fecal contamination of water. The organism causes primary amoebic meningoencephalitis, a disease characterized by sever frontal headache, nausea, fever, vomiting, and frequently, death. Most cases of this disease in humans have occurred after swimming in warm, fresh water. Swimming pools, mudholes, and mineral springs have all been implicated. Chlorine in 10 ppm is ineffective; however, salt contents of 0.7% are effective in controlling this organism.

Several other organisms have been implicated as being acquired via the fecal-oral route and could theoretically be acquired from wastewater effluent. However, their occurrence is rare. An attempt has been made to review the most important organisms and to bring attention to those which will be most frequently isolated.

SEVERITY AND FREQUENCY OF IDENTIFIED HAZARDS

The consequences from exposure to the hazards described above have occurred many times during recorded history. Prior to the use of disinfection and filtration in treating water supplies those hazards and consequences were major causes of human death and illness. But even today those consequences are occassionally realized. In order to provide a perspective of the potential problem, the larger, more recent outbreaks are described in the following discussion. The reported incidents involve the three fecal-oral pathways described above.

Hepatitis A.

During the period 1971-75, fourteen outbreaks of viral hepatitis affecting 368 people occurred which were associated with drinking water (Craun, et al., 1976). In 1975-1976, one outbreak occurred affecting 17 individuals (Craun, et al., 1979); and it also involved drinking water.

Salmonella typhimurium.

In 1965, in Riverside CA., more than 15,000 cases of salmonellosis caused by Salmonella typhimurium occurred due to a contaminated water supply that was not chlorinated. In 1978, 700-800 people in Suffolk County, NY became ill due to Salmonella typhimurium. Wastewater in a clogged slop sink in a catering facility was cultured and found to be positive.

Shigella.

In Dubuque, Ia., waterborne cases of Shigellosis were documented in 1974 (Rosenberg, et al., 1976). Thirty-one of 45 cases of Shigella sonnei were traced to swimming in water receiving wastewater effluent from the Dubuque treatment plant; however, state officials did not unequivocally identify the cause of the illness as the effluent from the wastewater treatment plant. The Dubuque wastewater was exposed to partial secondary treatment and chlorination; and, at the time of the outbreak, the effluent had fecal coliform counts up to 12,000,000/100 ml.

Enteropathogenic E. coli.

One thousand cases of diarrhea due to enteropathogenic E. coli occured at Crater Lake National Park in 1975. The water supply was identified as the source of the bacteria.

Vibrio cholerae.

A case of cholera occurred in Florida in 1980. The victim had eaten approximately six dozen raw oysters in a four day period. Two more cases of cholera followed this one, also due to eating raw oysters. In 1978, a case of cholera occurred in Louisiana due to ingestion of clams (Morb. Mort. Report, Dec. 19, 1980).

Campylobacter fetus sub jejuni.

As many as 2,000 out of 10,000 town residents experienced gastroenteritis in Vermont during a two week period in 1978 (Morb. Mort. Report, June 23, 1978). A strong association was found between illness and the consumption of water from the town water supply. The water was chlorinated but not filtered. Supplementary water sources were periodically used that were not chlorinated.

Yersinia enterocolitica.

In Europe, 10 out of 50 wells or non-chlorinated water works have been found to contain Yersinia enterocolitica (Stern and Pierson, 1979). In 1976, in Oneida City, NY, 220 school children became ill due to this organism. The mode of transmission was found to be chocolate milk. Many unnecessary appendectomies were performed. In 1972, an elderly man became ill during a hunting trip. The organism, Yersinia enterocolitica, was cultured from a mountain stream from which he had drunk. Infectious levels are not well established, but one individual in a human volunteer study did become ill when 3.5 billion cells of Yersinia were consumed (Stern and Pierson, 1979).

Giardia lamblia.

Outbreaks of Giardiasis occur most commonly in the Rocky Mountains, New England, and the Pacific Northwest. The largest waterborne outbreak of Giardiasis occurred in New York in 1974-75. An estimated 4,800-5,300 persons were affected. Disinfection, but not filtration, of the water supply was practiced. A resort town in Colorado which practices both filtration and chlorination had a Giardia outbreak in 1979. Giardia-like illness was described by both residents of the town and visitors to the town (Morb. Mort. Report, March 21, 1980).

Viruses.

In July 1979, 239 cases of gastrointestinal illness were reported among swimmers at a lake in Macomb County, Michigan. Subsequent sampling found no violations of bacteriological criteria for recreational waters so the etiologic agent was assumed to be viral (Morbidity and Mortality Weekly Report, Sept.7, 1979).

While the reported incidents provide a qualitative measure of the severity and frequency of the identified hazards the data are not sufficient to support quantitative assessment. This deficiency is even more obvious when the data are disaggregated by the pathways described above. Furthermore, only two of the reported incidents appear to have occurred as a result of exposure by swimming , and only one of those waters is known to have received a wastewater discharge. During the period 1971-1975, the major causes of outbreaks involving municipal water supply systems were deficiencies in the distribution system, some involving the influx of raw wastewater. For the period 1975-1976, the majority of outbreaks were caused by inadequately treated water. The causative agent for 55% of 223 outbreaks since 1971 is unknown.

Additional qualitative information is provided by studies focused on exposure via recreational waters and via occupation. The earliest study of significance was reported in 1953 (Stevenson, 1953), and this study concluded that the incidence of gastrointestinal symptoms was detectable among swimmers using fresh water with total coliform densities above 2,300 - 2,400 coliforms per 100 ml. Shortly thereafter, Moore (1959) reported no association between the incidence of polio or salmonellosis and swimming in polluted marine waters. These studies had diverse effects. The first study became the basis for most current coliform standards for recreational water, and the second study created the impression that swimming in sewage polluted seawater was not a health hazard. These studies have recently been reviewed in more detail by Cabelli (1981).

The incidence of gastrointestinal illness associated with swimming in marine waters of varying levels of pollution has recently been studied at three locations (Cabelli, 1981). In general, Cabelli found:

1. the incidence of enteric symptoms were higher for swimmers than non-swimmers,
2. only gastrointestinal symptoms increased with increasing levels of pollution,
3. rates of illness were higher for children than for adults,
4. symptoms declined with increased swimming time, and
5. in a body of water actually receiving raw wastewater, residents of the area who frequently visited the beach had much lower incidence of illness than visitors to the area, suggesting that acclimation is a factor.

Cabelli's work is discussed further in the dose-response section below.

Occupational exposure to wastewater was recently examined (EPA, 1980). The purpose of this study was to determine the effects of exposure of wastewater treatment plant workers to viruses, bacteria, and parasites in wastewater. Experienced and inexperienced workers and a control group not associated with treatment plants were studied. A total of 506 individuals were recruited for the study.

The study failed to demonstrate an increased risk to wastewater treatment plant workers due to bacterial, viral, and parasitic agents; however, gastrointestinal illness rates were higher in inexperienced workers. Bacterial cultures were done only for Salmonella and Shigella. Several other bacterial agents are described previously in this report which require special techniques for isolation. By such limited selection, it is possible that some disease causing organisms were overlooked.

Even with Cabelli's recent work, the relationship between the concentration of indicator organisms in recreation water and the incidence of illness is not well defined, especially in the fresh water environment. In this study the available information is used to illustrate a risk assessment methodology, but the reader is cautioned to assess any results within the constraints of the available information.

NO DISINFECTION RISK MODEL

A no-disinfection risk model must interrelate the probability of a consequence occurring with the quantity of hazardous organisms discharged, the dilution effect, the dieaway effect (or possibly, the regrowth effect), the potential insult to the recreational water user, and the relationahip between the magnitude of the insult and the realization of the consequences (the dose-response relationship). Each of these subjects is discussed below in the sequence given above, and the discussion ends with a synthesized model.

Magnitude of Discharge Hazards--

Conventional wastewater treatment provides reductions in microbes by way of the following processes: sedimentation, aeration and sedimentation, and natural dilution and die-off during surface water discharge. The typical levels of microorganisms entering a treatment plant are given in Table 16.

TABLE 16. INFLUENT CONCENTRATION RANGES FOR PATHOGENIC AND INDICATOR ORGANISMS (EPA,1979a)

Organism	Number/100ml	
	minimum	maximum
Total Coliforms	1,000,000	46,000,000
Fecal Coliforms	340,000	49,000,000*
Fecal Streptococci	64,000	4,500,000
Virus	0.5	10,000

* - Apparently, the samples containing the maximum fecal coliform levels were not analyzed for total coliforms because fecal coliform levels can never exceed the total coliform levels.

Table 17 shows the percent reduction of microorganisms by primary and secondary treatment. Reductions in bacteria and viruses up to 99% by primary and secondary treatment are cited in a recent EPA publication (EPA, 1979b). Presently, the quantitative measurement of fecal coliforms serves as an index to the presence of fecal contamination. Treatment of water, it is assumed, removes pathogens in proportion to the reduction in indicator organisms.

A relationship has been reported (Kerr and Butterfield,1943) between coliforms and typhoid bacteria in wastewater. The results were approximately 27.5 Salmonella sp. (Salmonella other than typhi) per 100,000 coliforms, and considerably fewer Salmonella typhi per 100,000 coliforms.

TABLE 17. MICROORGANISM REDUCTIONS BY CONVENTIONAL TREATMENT PROCESSES (Okun and Panghis, 1975; Craun, et al., 1976)

Microorganism	Primary treatment removal,%	Secondary treatment removal,%
Total coliforms	<10	90-99
Fecal coliforms	35	90-99
Shigella sp.	15	91-99
Salmonella sp.	15	96-99
Escherichia coli	15	90-99
Virus	<10	76-99
Entamoeba histolytica	10-50	10

Using this relationship and assuming median Table 17 reductions in microbes, the levels of microbes shown in Table 18 can be expected after secondary treatment.

TABLE 18. SECONDARY EFFLUENT RANGES FOR PATHOGENIC AND INDICATOR ORGANISMS

Organism	number/100ml	
	minimum	maximum
Total Coliforms	45,000	2,020,000
Fecal Coliforms	11,000	1,590,000
Fecal Streptococci*	2,000	146,000
Viruses	0.05	1,100
Salmonella sp.	12	570

* assuming removal efficiencies for fecal streptococci similar to the fecal coliform removal efficiencies.

Dilution Effect--

Dilution of a wastewater discharge will reduce the concentration of the hazardous organisms. This dilution effect can be incorporated into the model by multiplying the quantity of discharged organisms by a dilution factor which is defined for streams as the ratio of the discharge flow to the sum of the discharge and receiving stream flows. For lakes receiving discharge the dilution factor would be the ratio of the discharge flow to the lake outflow. This dilution factor will calculate an organism concentration for the lake that will then be diminished by the dieaway factor discussed below. The equilibrium concentration in the lake will then be a result of both dilution and dieaway.

Die-away of Pathogen Discharges--

The natural processes of die-off and inactivation will eventually reduce pathogen levels substantially. A bacterial die-off study has been reported (Dutka, et al., 1980) in Canada. A 200 ml aliquot of a four-day old Serratia culture was inoculated into a stream. The suspension contained approximately 100 million cells/ml. Serratia is a bacterium closely related to Klebsiella, a common isolate in normal human stools. The organism was recovered for a period of up to 22 days and traced for a distance of 20 kilometers (approximately 12.4 miles). In addition to the river studies, studies were done in lakes, and survival times up to 28 days were found for E. coli, enterococcus, and Salmonella thompson. When pollution levels in lakes were compared, it was noted that faster die-off times were observed in less polluted lakes. This probably occurs because there are less nutrients and organic carbon available in the less polluted bodies of water. The bacteria, therefore, are not able to reproduce at a rapid rate. Any inferences from a study of this type must consider the variations in numbers of microorganisms between treated wastewater effluent and a broth culture as utilized in the study. There are many competitive organisms in wastewater effluent, and nutrients vary from those supplied in a laboratory broth, depending on the nature of the discharge.

In arid and semi-arid areas, such as Denver, wastewater effluent may make up a large portion of stream flow. For example, in Clear, Sand, and Cherry Creeks in the Denver area, flow from wastewater may constitute 40-70% of stream flow (EPA, 1977). Dilution effects in this case are minimal, and die-away will be the primary mechanism reducing pathogen populations.

Numerous mathematical models have been proposed for modelling bacterial die-away; however, models based on first order kinetics are most frequently selected for application. The first order model for die-away in streams is:

$$N = N_o \, e^{(-kt)}$$

where N_o = the initial concentration of microbes discharged into the stream, and

N = the concentration of the microbes t time units after discharge into the stream.

A first order model for die-away in standing water bodies (i.e. lakes) is:

$$N = N_o/(1+kt_d)$$

where N_o = the concentration in microbes in the water body's inflow,

N = the concentration of microbes in the water body's discharge, and

t_d = the hydraulic detention time in the water body based on the water body's discharge.

The rate constant k can be determined from a dieaway study of typical lakes or streams in a planning area. For example, if a summer dieaway stream study found a 30% reduction of the microorganisms in two days, k would be:

$$k = (1/t) \ln (No/N)$$

$$k = (1/2) \ln (100/70)$$

$$k = 0.18 \text{ per day.}$$

Rate constants may also be found in the literature (Bitton,1978 and Berg, 1978).

Potential Insult--

The phrase "potential insult" refers to the potential for a human to ingest pathogenic organisms. The method of expressing this factor will depend on the type of dose-response model used. For example, if the dose-response model is defined as the number of organisms required to elicit a response in a given fraction of insults then the potential insult must be stated in terms of total organisms ingested. This quantity can be calculated by multiplying the organism concentration by the amount of water ingested while swimming which in this study was assumed to fall in the 50 - 500 ml range.

On the other hand, a dose-response model may relate probability of illness to organism concentration. In that case, the potential insult is incorporated into the dose-response model so this element does not have to be estimated separately.

Dose-Response--

Data relating dosage levels of microbes to risk of disease has been available for only the last few years. Available data seem to indicate that a considerable number of organisms are required to elicit a response for several pathogens. For example, the most severe of all diarrheal diseases, cholera, is reported to require 100,000,000 organisms to elicit a response, and of those challenged, only 1-10% will develop clinical manifestations (Sack, et al., 1980). On the other hand, it has also been reported that 10-200 Shigella can elicit a response in those challenged (Sack, et al., 1980). More data collection needs to be done on Shigella since this is the bacterial pathogen which apparently requires the lowest number of ingested organisms to initiate a disease process.

The infective dose for pathogenic protozoa is reported to be small in some cases. For example, one Giardia cyst has been reported to be infective. However, in an experiment where adult humans were challenged with ten organisms, 76-100% did not become ill (EPA, 1979a). The infective dose for Entamoeba histolytica is likewise not well established.

Knowledge concerning the infective dose of viruses is perhaps the most poorly established. Many virologists believe that one active virus is sufficient to initiate a response (Mahdy, 1979).

Two dose-response models were found in the literature for Salmonella (Mechalas, et al., 1972; EPA, 1979b). Both models are represented graphically in Figure 4, and the lack of agreement between the two models is substantial and obvious. For example, 10^5 Salmonella sp. organisms per liter causes a risk of 1 illness in 5 billion exposures (EPA, 1979b), or a risk of 1 illness in 3300 exposures (Mechalas,1972). However, the levels of Salmonella in secondary treatment effluent, as shown in Table 18, would seem to pose a low risk of illness.

In order to use a dose-response model with water quality data as an input, the dose units should be indicator organisms instead of pathogens. The Mechalas (1972) models include coliform and fecal coliform curves in addition to the Salmonella and virus curves. The implication in those models that the ratios between the indicator species and the pathogens are constant is debatable and raises doubt about the models' accuracy.

Because of the two problems cited above with the reported Salmonella models, those models were not used in the no disinfection risk model.

Cabelli (1981) has also presented quantitative regression models relating the occurrence of gastrointestinal symptoms per 1000 swimmers to the concentration of an indicator organism. His work also evaluated several indicator organisms, and he found far better correlation using enterococcus or E. coli than using the more commonly used coliform and fecal coliform indicator organisms. In fact, the correlation for the coliform indicators are so poor that a useful symptom-coliform model cannot be synthesized. The dependence of the recommended Cabelli model on Enterococcus input-data greatly diminishes the model's usefulness

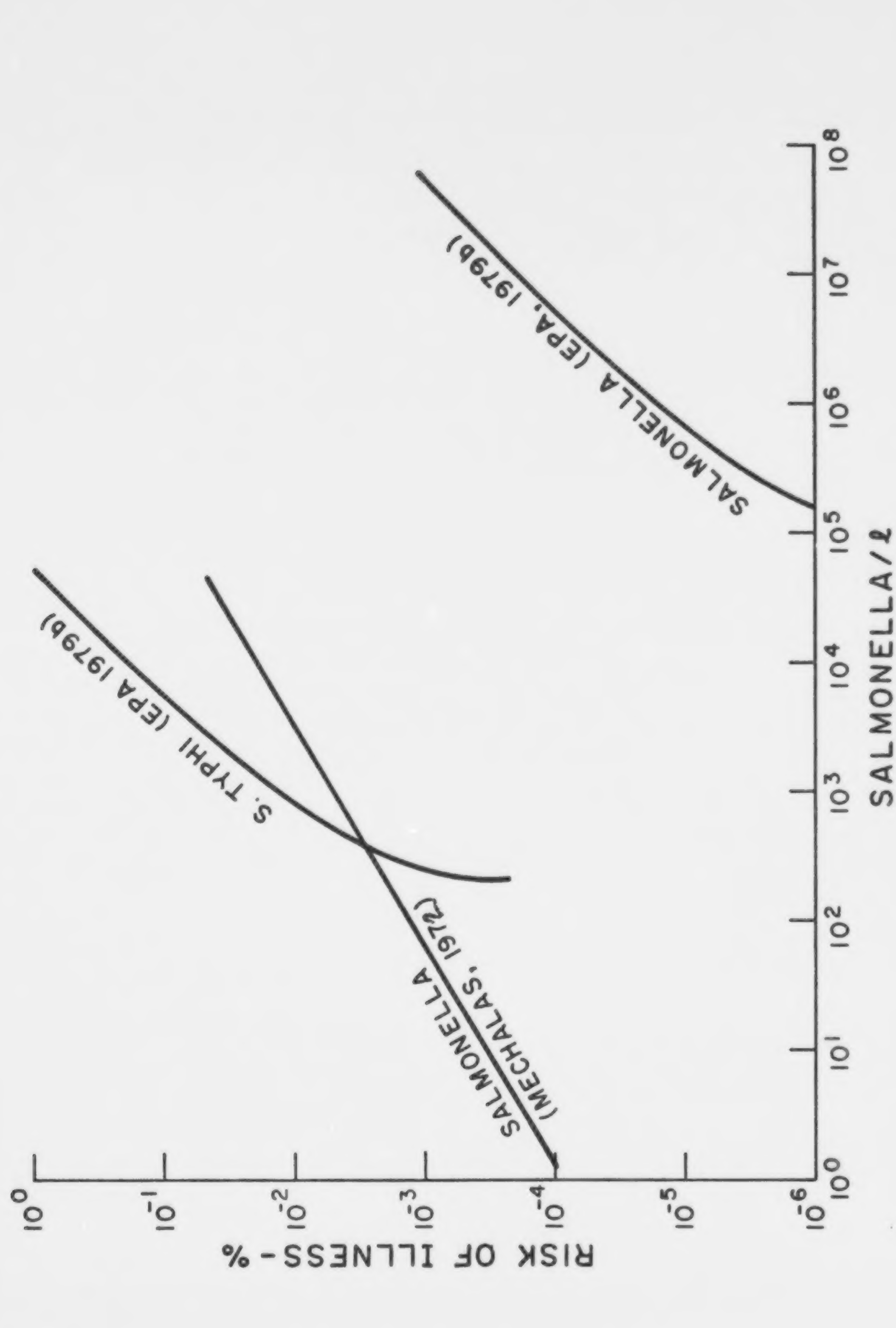

FIGURE 4. COMPARISON OF SALMONELLA DOSE-RESPONSE MODELS

since very little *Enterococcus* data are available. Cabelli presents several regression models, and the model best suited to this study's approach is:

$$y = 11.85 \log x - 0.58$$

where y = the number of swimmers developing gastrointestinal symptoms per 1000 swimmers and

x = the mean enterococcus density per 100 ml.

Assembled Model--

The relationships presented above can be assembled into the following mathematical models. Using Cabelli's dose-response model and assuming the wastewater is discharged to a stream the model becomes:

$$y = 11.85 \log[A\ Qe \exp(-kt)/(Qe + Qs)] - 0.58$$

where y = the number of gastrointestinal symptoms expected per 1000 swimmers,

A = the enterococcus concentration in the wastewater discharge in organisms/100 ml,

Qe = wastewater flow rate in volume per unit time,

Qs = receiving stream flow rate in the same units as Qe,

t = time of travel in the stream from the point of discharge to the point of exposure in time units, and

k = the rate constant as described above with the same units as t to the minus 1 power.

The model for discharge to a standing water body would be:

$$y = 11.85 \log[A\ Qe/(Qd(1 + kt_d))] - 0.58$$

where Qd = standing body discharge flow rate in the same units as Qe,

t_d = hydraulic residence time in the water body, and

y, A, Qe, and k are defined above.

Probability factors can be added to the model by estimating probabilities for the input variables and then integrating them with the Cabelli model's statistics. The use of the model shown above is illustrated by an example in Section 8.

SECTION 8

ENERGY AND COST CONSIDERATIONS

ENERGY

Chlorination

The on-site energy requirements for the chlorination of wastewater are small compared with those of ozonation or ultraviolet irradiation. An analysis of energy requirements for chlorination of municipal water supplies has recently been published by Clark (1981). In this analysis of chlorination energy demands, values for the energy efficiency, in kWh/kg Cl_2, should vary little between wastewater and drinking water plants of the same size. Hence, the energy requirements listed in Clark's paper will be used as a good approximation for the energy efficiency of the chlorination process in wastewater treatment plants. Clark reports that the energy efficiency is a function of the disinfectant capacity of the plant. For example, the energy efficiency is given as 1.88 kWh/kg Cl_2 at a design capacity of 37.9 kg Cl_2/d, and a value of 1.41 kWh/kg Cl_2 for a design capacity of 75.7 kg Cl_2/d. Doses in the range 4 - 16 mg Cl_2/L are typical of wastewater treatment plants. For a dose of 6 mg/L, assuming 100% Cl_2 absorbed, a 3790 m^3/d plant would require 22.5 kg Cl_2/d, i.e., $(3790\ m^3/d) x (6 mg/L) x (kg/10^6\ mg) x (10^3\ l/m^3) = 22.5$ kg Cl_2/d. Based upon the analysis of Clark, (Table 10, Clark, 1981), a plant with a capacity of 22.5 kg Cl_2/d would have an energy efficiency of about 3.7 kWh/kg Cl_2.

Only on-site energy requirements are included in this study. Off-site power consumption associated with chlorine manufacture and the energy involved in transport are not included in energy calculations. This approach results in an apparent lower energy cost for chlorine as compared with alternative disinfectants that must be produced on-site.

Ozonation

In this section the energy requirements for wastewater disinfection by ozonation are discussed. Energy-intensive steps involved in ozonation are discussed first, followed by some comments on how the quality of the influent may affect the energy requirements for ozonation of water. Energy data for ozonation of wastewater and municipal water supplies will be presented and compared, in the context of differences in water quality, as well as the variance associated with different equipment. This analysis will be used to provide a range of energy values, in kWh/kg O_3, for ozonation of wastewater. Finally, an estimate of required kWh per unit of treated wastewater will be obtained for a typical range of values for the mass transfer efficiency.

Energy-Intensive Steps in the Ozonation Process--

Though air is by far the most common feed gas for ozonators treating municipal water supplies, in wastewater treatment plants in the U.S (those either operating or in various stages of development) enriched oxygen (air enriched by oxygen) is roughly as common a feed gas as air. In wastewater treatment, where oxygen may be generated on-site for oxygen-activated sludge, the ready availability of oxygen as a feed gas offers the possibility of higher levels of ozone production for the same energy input to the ozonating equipment. However, if the feed gas is air or partially-enriched oxygen (associated with an ozonation process in which oxygen is recycled), air pretreatment, involving compression, refrigeration, and drying, is essential (Rosen, 1976).

Manufacturers of ozone generators may sometimes promote values for energy requirements obtained under the most ideal conditions. However, the energy efficiency of a specific ozone generator is a function of many variables, including: power level, condition of the generator, cooling fluid temperature, as well as the flow rate, moisture content, and oxygen content of the feed gas. Of course, the energy efficiency will also vary for different model generators. The effect of these variables on energy efficiency of ozone generation has been discussed in detail by Carlins (1981).

In addition to the energy cost of feed gas pretreatment and ozone generation, the overall energy efficiency will depend largely on the mass transfer efficiency of the contactor, as well as the dosage level required for adequate disinfection. Finally, additional energy is usually expended to operate an ozone destruct unit which prevents the contactor off-gases from raising ambient ozone levels beyond acceptable levels.

Qualitative Comparison of Energy Requirements for Ozonation of Wastewater and Potable Water--

The rather limited energy data available for wastewater treatment plants using ozone suggest that the much larger body of published data for the treatment of municipal water supplies should also be considered. The energy efficiencies, in kWh/kg O_3, should be very similar; however, some cautionary observations are appropriate. First, the dosage levels required for adequate disinfection are higher in wastewater treatment, not only because of the presence of higher levels of microorganisms but also the presence of higher levels of ozone demand. The presence of various organic and inorganic reactants has led to a second problem. The mass transfer efficiency at some of the early U.S. wastewater treatment plants has turned out to be considerably lower than expected. This is likely due, in part, to the then embryonic state of development of the ozonation process in wastewater treatment. The use of PVC piping and inappropriate epoxies has been associated with serious leaking in some contactors (Jain, et al., 1979; Rakness and Hegg, 1980) which would cause low transfer rates. Moreover, there is some evidence that the mass transfer capabilities of contactors are affected by the level of reactant pollutants (Rakness and Hegg, 1980). Thus, care must be taken that in a laboratory pilot study of a particular diffuser design, the water quality be as close as possible to that at the proposed plant. A low mass transfer efficiency, associated with an under designed or poorly designed contactor, implies a higher value of kWh/unit treated water, as well as higher levels of ozone in the contactor off-gases which enter the destruct unit. Finally, ozonation systems used in the treatment of municipal water supplies use air as a feed gas almost exclusively, so the data from such systems will have to be adjusted appropriately for comparison to wastewater plants which often use oxygen as a feed gas.

Energy Efficiency (kWh/kg O_3) for Ozonation of Wastewater and Potable Water--

Air as Feed Gas.--Table 19 presents the energy efficiency, in kWh/kg O_3, for twelve European and Canadian plants treating potable water. The data were published in a 1978 EPA report (Miller, et al., 1978) which included the results of a questionnaire mailed to numerous municipal treatment plants throughout Europe and Canada. In Table 19 only the data from those plants which provided a breakdown of energy consumption for various stages of the ozonation process are presented. The average values for air preparation, generation, contacting, and ozone destruct are, respectively, 7.8, 19.3, 6.6, and 8.7 kWh/kg O_3. The average total energy efficiency is 29.5 kWh/kg O_3, where some of the plants did not report energy consumption values for contacting or ozone destruct. Note that while the absence of an additional energy requirement for the contactor is consistent with a system driven by the positive pressure of the off-gases from the generator, a destruct unit would require additional power. An examination of the data in Table 19 reveals no clear pattern associated with an economy of scale; of course, various "noise" factors discussed above may effectively mask such a pattern.

Comparable data for wastewater treatment plants is quite sparse. Many U.S. plants have had severe difficulties getting up to full operational capability, with shake-down periods commonly

TABLE 19. ENERGY UTILIZATION FOR EUROPEAN AND CANADIAN PLANTS TREATING MUNICIPAL WATER SUPPLIES (Miller, et al.,1978); AND ONE U.S. PLANT TREATING MUNICIPAL WASTEWATER (Rakness and Hegg, 1980)

Plant	Capacity	Air Prep.	Ozone Gen.	Ozone Contact	Ozone Destruct	Total
	kg O_3/d	kWh/kg O_3				
Lock Turret	168	2	22.6	6.6	5.3	37
Durleigh	192	12	18	6		36
Cantineweg	120	3	21	0.6		24.5
Linz	27	14	21	10		45
Biel	130	2.3	17	3.7		23
Toulon	36	11	26		12	47
Villeneuve-la Garonne	96	12	18			30
Flins	264	12	18			30
Drummondville	41	2.2	15.4	4.4		22
Ile Perrot	9	9	21	15		45
Pierrefords	250	6	12			18
Sherbrooke	163	7.9	21.2			29.1
AVERAGES		7.8	19.3	6.6	8.7	29.5
Estes Park, CO	25.3	17.3	17.3	0	4.3	38.9

extending several months or a few years beyond the scheduled start-up date. These delays have been due to ozone-related problems, as well as other factors. However, significant energy data have been published for the Upper Thompson Sanitation District (UTSD) wastewater treatment plant in Estes Park, Colorado. Data for the UTSD plant at 70% of actual capacity, presented in Table 19, are in good general agreement with the energy efficiency of the European plants. The designers of the UTSD plant have noted a significant difference between the manufacturer's specifications for energy efficiency and actual operational values (Rakness and Hegg, 1980). They attribute this discrepancy to a faulty dew point measuring device which caused the moisture of the generator feed air to exceed the manufacturer's minimum specified value. They also note that if the UTSD ozone generator had achieved rated energy efficiency, the total energy efficiency would have been about 34 kWh/kg O_3 at 70% capacity, instead of the observed value of 38.9 kWh/kg O_3. Their projected value brings the UTSD data into even closer agreement with the average total energy efficiency of 29.5 kWh/kg O_3 for the European and Canadian plants.

O_2 as Feed Gas.--As discussed above, there are very limited energy utilization data for wastewater treatment plants using air as feed gas for ozonation; however, even less data are available for systems using oxygen as the feed gas. The Southwest Wastewater Treatment Plant in Springfield, Missouri, has been operating successfully for about three years, with careful monitoring of costs (Letterman,1981) and is the only source of energy data for oxygen-fed ozonation found in the literature. Oxygen is generated on-site for an oxygen activated sludge system, as well as for ozone generation. The Springfield system uses a once-through oxygen process, in which the feed gas is 98% pure oxygen, the generators provide a 3% conversion of oxygen to ozone, and off-gases are channeled directly to the biological system. There is no ozone destruct unit. Ozone mass transfer efficiency has typically been in the range of 80 -90%. Table 20 summarizes the energy requirements of the Springfield plant. The energy requirement for oxygen generation, 11 kWh/kg O_3, has been obtained from the reported values of .33 kWh/kg O_2 produced (Breitback,1981) and the 3% conversion efficiency for ozone generation from oxygen. The energy efficiency of the Springfield ozone generators, 10.9 kWh/kg O_3, is consistent with the energy efficiency for air-fed systems (Table 19) and the approximate two-fold increase in efficiency associated with oxygen-fed systems (Rice, 1980).

TABLE 20. ENERGY UTILIZATION FOR AN OXYGEN FED WASTEWATER OZONATION PLANT (Letterman, 1981)

Plant	Capacity	O_2 Generation	Blowers & Mixers	Generators	Total
	kg O_3/d		kWh/kg O_3		
Springfield MO	1360	11	0.53	10.9	22.4

Energy Utilization in kWh/Unit treated water--

The energy utilization, in kWh/unit treated water, will be a function of energy efficiency (kWh/kg O_3), the mass transfer properties of the system for ozone, and the water quality. In quantitative terms,

$$\text{Energy utilization(kWh/L)} = \text{energy efficiency(kWh/kg } O_3) \times \text{dosage(kg } O_3\text{/L)} / \text{mass transfer efficiency}$$

Doses in the range of 4 - 8 mg O_3/L are typical of plants which do not have significant amounts of industrial wastes in their influent waters (Rice, 1980). Table 21 provides energy utilization values, in kWh/L treated water, corresponding to an energy efficiency of 22, 30 or 35 kWh/kg O_3, a mass transfer efficiency of 50% or 85%, and an ozone dose of 1 mg/L.

TABLE 21. ENERGY UTILIZATION PER UNIT OF OZONATED WASTEWATER
kWh/l treated water/(mg O_3 absorbed/l)

Energy Efficiency kWh/kg O_3	Mass Transfer Efficiency 50%	Mass Transfer Efficiency 85%
22	4.4×10^{-5}	2.6×10^{-5}
30	6.0×10^{-5}	3.5×10^{-5}
35	7.0×10^{-5}	4.1×10^{-5}

Use of Table 21 is straight forward. For example, at 30 kWh/kg O_3, 85% mass transfer efficiency, and an absorbed ozone dose of 6 mg/L, a 3,785 m^3/d plant would require ((3,785,000 l/d) x (3.5×10^{-5} kWh/l/(mg/L)) x 6 mg/L =) 795 kWh/day.

Ultraviolet Radiation

Since the early 1900's ultraviolet light has been used, on occasion, to disinfect small quantities of drinking water (Scheible, 1980). The lack of residual disinfectant in the treated water, though generally viewed as a disadvantage for the treatment of potable water, may be an advantage for wastewater treatment. Various small pilot studies have explored the practicality of using UV for the treatment of wastewater. The first full-scale ultraviolet disinfection field study was conducted during a 13-month period in 1978-1979 at the Northwest Bergen County Water Pollution Control Plant in Waldwick, New Jersey (Scheible, 1980). The UV apparatus at the plant was capable of treating the full plant flow of 15,000-30,000 m^3/d (3.9-7.9 mgd). Plant data were used to estimate energy requirements for typical wastewater treatment plants. Energy requirements of 4.4×10^4 kWh/yr, 4.4×10^5 kWh/yr, and 4.4×10^6 kWh/yr were estimated for plants with design capacities of 1 mgd, 10 mgd, and 100 mgd, respectively. These values imply an energy utilization of 3.2×10^{-5} kWh/L of treated water. These values for the energy requirements and energy utilization are based upon doses required to meet current effluent standards. Existing standards do not include consideration of repair phenomena which may occur in bacteria subsequent to disinfection. It has been shown that the repair of bacterial DNA damaged by UV irradiation is enhanced if the organisms are subsequently exposed to visible light. The increase in dose required to compensate for the photoreactivation reaction would depend upon the season and geographical location of the plant. A factor of 2x increase in dose was used by Scheible(1980) as an approximation of the dose needed to compensate for photoreactivation which would increase the energy utilization to 6.4×10^{-5} kWh/l of treated water.

Comparison of On-Site Energy Requirements

An example comparison of the on-site energy requirements for the alternative disinfection processes considered can be developed using a 3790 m^3/d wastewater treatment plant. For wastewater treatment, absorbed doses in the range of 4-8 mg O_3/L or 4-16 mg Cl_2/l are typical of plants which do not have significant amounts of industrial wastes in their influent waters. For the example comparison, absorbed doses of 6 mg O_3/L and 10 mg Cl_2/l are assumed to be equivalent for the purposes of disinfection. An ozone transfer efficiency of 85% is also assumed. As discussed above, the energy efficiency for air-fed ozonation is about 30-35 kWh/kg O_3 for a wide range of plant sizes. For the sake of comparison, a value of 35 kWh/kg O_3, suggested by the data obtained from the 1.3 mgd UTSD plant in Estes Park, Colorado will be assumed. The energy utilization of oxygen-fed ozonation plants is assumed to be 22 kWh/kg O_3. The energy requirements, based on the above assumptions, for a 3790 m^3/d plant are shown in Table 22.

TABLE 22. EXAMPLE COMPARISON OF ENERGY REQUIREMENTS FOR ALTERNATIVE DISINFECTANTS
kWh/d

Disinfection Alternative	Energy Requirement kWh/d
Chlorination	71
Ozonation	
Air-fed	932
Oxygen-fed	586
Ultraviolet	
w/o photoreactivation	121
w/ photoreactivation	242

As discussed above, the energy requirement for chlorination will vary with treatment plant size. For smaller plants the requirement could be substantially larger than shown in Table 22, and for larger plants the requirement will decline slightly from the value shown.

COSTS

This section discusses capital and O & M costs for disinfection by chlorination and ozonation and reduces them to costs per 1,000 cubic meters. Sensitivity to some of the variables is estimated.

Chlorine

Capital Costs for Chlorine Facilities--

The 1980 EPA study of wastewater treatment construction costs identified 92 construction projects that included chlorination for disinfection. After adjusting all prices to the fourth quarter 1978, cost ($ million) was regressed on design flow (in mgd) to produce the following estimating equation for cost as a function of quantity (EPA FRD-11, 1980):

$$\text{Cost} = (6.33 \times 10^4) \times (Q^{0.65})$$

In order to bring this equation up to date, inflation to the first quarter of 1981 must be added. The EPA small city conventional treatment plant cost index for 1 mgd and 10 mgd chlorination systems and the large city advanced treatment index for the 100 mgd plant were used to add the inflation. The relevant indices are shown in Table 23 (EPA, 1981).

TABLE 23. CONSTRUCTION COST INDICES

PLANT SIZE m^3/day	1978 INDEX	1981 INDEX	PERCENT CHANGE %
3790	152.0	174.7	15.0
37900	152.0	174.7	15.0
379000	157.0	191.2	21.8

The wide variations in percent increase do not actually reflect differing inflation rates, but rather, a peculiarity in the base years. The 1981 figures are national averages while the 1978 figures are based only on data from Kansas City and St. Joseph, MO. The Kansas City data were within 1% of the national average in 1978, but the St. Joseph data was 5% above the average.

Non-construction project costs (design costs) also need to be added to costs. Administration, legal costs, architects and engineering fees, inspections, and contingencies add another 28%

to new projects or 36% on enlargement or upgrading projects (EPA, FRD-11, 1980). A median figure of 32% for non-construction costs is used in this analysis.

Capital costs shown in Table 24 for a chlorine disinfection system are the product of the basic estimate, inflation, and the non-construction cost ratio.

TABLE 24. CHLORINATION CAPITAL COSTS

PLANT SIZE m^3/day	1978 COSTS $	INFLATION COSTS $	DESIGN COSTS $	1981 COSTS $
3790	63,000	9,450	23,550	96,000
37900	294,000	44,100	107,900	446,000
379000	1,367,000	298,000	533,000	2,198,000

The peculiarities of individual cases lead to large variations around these averages. The EPA data on individual construction jobs show variations by more than a factor of three both up and down in about 20% of their cases. Area costs of construction vary from the average by up to 20% even if the highest and lowest cost cities (New York and Charlotte, NC) are excluded.

Annual costs for several interest rates with twenty year amortization periods are shown in Table 25 for three sizes of treatment plants. In order to estimate annual costs it is necessary to select an amortization period, an amortization

TABLE 25. ANNUAL CHLORINATION CAPITAL COSTS

INTEREST RATE %	AMORTIZATION PERIOD yrs	PLANT SIZE m^3/d 3790	37900	379000
0	1	$96,000	$446,000	$2,198,000
0	20	4,800	22,300	109,000
3	20	6,432	29,882	147,226
6	20	8,352	38,802	191,226
9	20	10,464	48,614	239,582
12	20	12,864	59,764	294,532
15	20	15,360	71,360	351,680

interest rate, and a utilization rate. The latter factor increases the cost per unit of wastewater treated because of unused capacity.

A twenty year amortization period is assumed for this analysis. The interest rate on good quality municipal bonds was approximately 11% in mid 1981. The rate is held down by the federal and state government tax exemption, and it also fails to fully reflect increasing risks as communities approach the limits to their bonding capacities. On the other hand, these rates are pushed abnormally high by the fear of inflation and the federal effort to end inflation. Nine percent (9%) was selected as a reasonable interest rate for amortizing a wastewater investment in this analysis. Other interest rates might vary the amortization per year by up to a factor of two.

Unit capital costs for chlorination are shown in Table 26 for three utilization rates. The annual amortization costs for 9% interest and a 20 year amortization period were used as bases for the unit costs shown. Cost variations from interest rates and regional differences in construction costs would scale this whole table upward or downward proportionately. For example, a six percent interest rate would reduce all capital costs by 20 percent.

TABLE 26. UNIT CHLORINATION CAPITAL COSTS

PLANT SIZE m^3/day	ANNUAL COST $	UTILIZATION RATE %	ANNUAL UNIT COST $/1000 m^3
3790	10,464	100	7.56
		80	9.46
		60	12.61
37900	48,614	100	3.51
		80	4.39
		60	5.86
379000	239,582	100	1.73
		80	2.16
		60	2.89

Operating and Maintenance (O&M) Costs Using Chlorine--

The O & M costs for disinfection by chlorine can be categorized as chemical, labor, supplies, and power. Chlorine alone will typically account for half or more of all O & M costs.

Chlorine dose can vary with the wastewater being treated, and a common figure in the literature appears to be 10 mg/L (Opatken, 1978). The 1980 WPCF survey suggests a median dose of just over 6 mg/L (WPCF, 1980). The costs per unit of treated wastewater for these two dosages are shown in Table 27.

TABLE 27. CHLORINATION DISINFECTANT COSTS

PLANT SIZE m^3/day	PRICE $/kg	DOSE mg/L	ANNUAL USAGE kg/yr	UNIT COST $/1,000 m^3
3790	0.24	6	8,300	1.44
		10	13,800	2.40
37900	0.24	6	83,000	1.44
		10	138,000	2.40
379000	0.18	6	830,000	1.08
		10	1,380,000	1.80

Price for chlorine also varies. In the Denver area, railroad tank car lots can be bought for $0.18/kg (Metropolitan Denver Sewage District No.1) and 908 kg cylinders for about $0.24/kg (Denver Water Dept.). Smaller (68 kg) cylinders appear to cost more than twice as much and are probably not cost effective even for a 3790 m^3/d plant. Median price for chlorine in the 1980 WPCF survey was $0.26/kg (WPCF, 1980). The unit costs shown in Table 27 are based on the assumption that the smaller plants will use 908 kg cylinders and the large plant will purchase chlorine in railroad tank cars.

Labor is the second category of operating cost. Maintenance, labor and supervision are assumed to be proportional to plant investment totaling 1% of cost. Labor cost is assumed to be $10 per hour. Labor costs per unit of wastewater treated are shown in Table 28.

TABLE 28. CHLORINATION LABOR COSTS

PLANT SIZE m^3/day	ANNUAL LABOR hrs	ANNUAL COSTS $	UNIT COSTS $/1,000 m^3
3790	460	4,600	3.33
37900	1,840	18,400	1.33
379000	7,360	73,600	0.53

Power costs are primarily for heating, lighting and ventilation. While electricity can still be bought at 4-5 cents per KWh, new capacity generally costs at least 6 cents and in some cases more. Power costs per unit of wastewater treated are shown in Table 29.

TABLE 29. CHLORINATION POWER COSTS

PLANT SIZE m^3/day	ANNUAL CONSUMPTION kWh	ANNUAL COSTS $	UNIT COSTS \$/1,000 m^3
3790	10,000	600	0.43
37900	20,000	1,200	0.09
379000	20,000	1,200	0.01

Supplies for maintenance are estimated at 1% of capital costs.

Total O & M cost using chlorine for disinfection are the sum of the components. At 3790 m^3/d, these costs are most sensitive to labor costs. Sensitivity to the cost of chlorine is dominant in the larger plants. Chlorination operating and maintenance costs are summarized in Table 30.

TABLE 30. CHLORINATION O & M COSTS SUMMARY \$/1,000 m^3

	PLANT SIZE m^3/day		
	3790	37900	379000
Disinfectant	1.44 - 2.40	1.44 - 2.40	1.08 - 1.80
Labor	3.33	1.33	0.53
Power	0.43	0.09	0.01
Supplies	0.08	0.04	0.02
Totals	5.28 - 6.24	2.90 - 3.86	1.64 - 2.36

As with capital costs, O & M costs per unit actually disinfected can be expected to increase with underutilization. The effects of underutilization on chlorination O & M costs is shown in Table 31 with the labor costs per unit of wastewater treated assumed to be constant.

TABLE 31. UTILIZATION EFFECTS ON CHLORINATION O & M COSTS
$/1,000 m^3

PLANT SIZE m^3/day	PERCENT UTILIZATION 60	80	100
3790	6.58 - 8.18	5.77 - 6.97	5.28 - 6.24
37900	3.95 - 5.55	3.29 - 4.49	2.90 - 3.86
379000	2.38 - 3.58	1.92 - 2.82	1.64 - 2.35

Chlorination Costs Summary--

Total chlorine disinfection costs are summarized in Table 32. The chlorination costs shown are fairly consistent with similar costs cited in the literature. Van Note (1978), Opatken (1978), and Gupta (1976) all show higher O & M costs after adjusting to 1981 prices because they used higher chlorine prices. Opatken also assumes a higher labor input than used in this analysis. Opatken and Gupta estimate costs for 1.3 mgd and 2.0 mgd plants, respectively, and apparently used the more expensive 68 kg cylinders. The estimates of capital cost amortization by Van Note, Opatken and Gupta are lower than the estimate in this analysis because they used a lower interest rates. Gupta's lower capital costs are offset by his inclusion of additional capital for dechlorination and post-aeration. The differences reflect primarily the differences in conditions when the studies were done. A fourth study by Nall (1980) did not break down the costs, but found total chlorine disinfection costs similar to the results in this analysis for the small plant and somewhat lower costs for the two larger plants.

TABLE 32. CHLORINATION COSTS SUMMARY
$/1,000 m^3

PLANT SIZE m^3/day	PERCENT UTILIZATION 60	80	100
3790	19.19 - 20.79	15.23 - 16.43	12.84 - 13.80
37900	9.81 - 11.41	7.68 - 8.88	6.41 - 7.37
379000	5.27 - 6.47	4.08 - 4.98	3.37 - 4.08

Notes - minimum unit cost corresponds to 6 mg/L Cl_2 dosage
- maximum unit cost corresponds to 10 mg/L Cl_2 dosage
- interest rate = 9%
- amortization period = 20 years

Ozone

Capital Costs for Ozone Facilities--

Ozone cost experience in this country is extremely limited; however, in Europe, there is substantial experience with drinking water treatment rather than wastewater. As a result, cost estimates for ozone treatment of wastewater have not been confirmed by substantial experience and are subject to a wider band of uncertainty than chlorine disinfection costs.

The major controlling factor for ozone disinfection cost is the amount of ozone that must be applied to the wastewater. This in turn is the product of two factors, the amount needed for disinfection and efficiency with which an applied dose becomes available.

The amount of ozone required for disinfection will, of course, vary with the wastewater and the standards for disinfection. Venosa, et al.(1978) found that ozone doses of 4 mg/L of wastewater were sufficient to meet EPA standards of disinfection with reasonably good secondary effluent, and 5 mg/L of water provided sufficient safety margin.

The Venosa experiments found absorption rates between 50 and 90 percent for ozone (Opatken, 1978). Operators of ozone disinfection plants report absorption rates as low as 40 percent. Ozone generation requirements for a 5 mg/L dose at varying absorption rates are shown in Table 33.

TABLE 33. OZONE PRODUCTION REQUIRED TO PRODUCE AN EFFECTIVE DOSE OF 5 MG/L
kg/day

PERCENT ABSORBED %	APPLIED DOSE mg/L	PLANT SIZE, m^3/d		
		3790	37900	379000
90	5.55	21	209	2092
70	7.14	27	268	2679
50	10.00	38	377	3768
40	12.50	47	472	4722

In 1978, EPA published construction costs for ozone generation and contactor systems using an air feed for the smallest systems and pure oxygen for larger systems (Gumerman, 1978). Ozonation capital costs with design and inflation costs included are shown in Table 34. The minimum cost shown corresponds to a contactor efficiency of 90%, and the maximum cost corresponds to a contactor efficiency of 40%.

TABLE 34. OZONATION CAPITAL COSTS

PLANT SIZE m^3/d	MIN or MAX	1978 COSTS $	INFLATION COSTS $	DESIGN COSTS $	1981 COSTS $
3790	min	130,000	19,500	47,800	197,300
	max	230,000	34,500	84,700	349,200
37900	min	600,000	132,000	234,000	966,000
	max	1,000,000	220,000	390,000	1,610,000
379000	min	2,800,000	616,000	1,093,000	4,509,000
	max	4,300,000	946,000	1,679,000	6,925,000

Ozonation capital costs are two to four times higher than chlorination capital costs depending on the ozone contactor's efficiency. The estimates in Table 34 assume the small plant will use an air-fed ozonation system and the two larger plants will use an oxygen-fed system. Ozone production from air reportedly requires about fifty percent more capital than ozone production from oxygen (Nall, 1980) per unit of ozone produced.

Amortized ozonation capital costs are shown in Table 35 for several interest rates and amortization periods.

TABLE 35. ANNUAL OZONATION CAPITAL COSTS

INTEREST RATE %	AMORT. PERIOD yrs	MAX or MIN	PLANT SIZE m^3/d		
			3790	37900	379000
0	1	min	$197,300	$966,000	$4,509,000
		max	349,000	1,610,000	6,925,000
0	20	min	9,900	48,000	225,000
		max	17,000	80,000	346,000
3	20	min	13,000	65,000	302,000
		max	23,000	108,000	464,000
6	20	min	17,000	84,000	392,000
		max	30,000	140,000	602,000
9	20	min	21,000	105,000	492,000
		max	38,000	175,000	754,000
12	20	min	26,000	129,000	604,000
		max	47,000	216,000	927,000
15	20	min	32,000	155,000	722,000
		max	56,000	258,000	1,107,000

The annual ozonation capital costs per unit of wastewater treated are shown in Table 36. The unit costs shown are based on an assumed interest rate of 9% and an amortization period of 20 years. The effects of underutilization on the unit costs is also shown.

TABLE 36. OZONATION UNIT CAPITAL COSTS

PLANT SIZE m^3/d	ANNUAL COST $	UTILIZATION RATE %	ANNUAL UNIT COSTS $/1,000 m^3
3790	21,000 - 38,000	100	15.18 - 27.47
		80	18.98 - 34.34
		60	25.30 - 45.78
37900	105,000 - 175,000	100	7.59 - 12.65
		80	9.49 - 15.81
		60	12.65 - 21.08
379000	492,000 - 754,000	100	3.56 - 5.45
		80	4.45 - 6.81
		60	5.93 - 9.08

Operating and Maintenance Costs for Ozonation--

Ozone operating costs differ from those of chlorine disinfection primarily in that the chlorine cost is replaced by power for producing ozone. Opatken (1978) estimated power needed for ozone production from air to be 30.8 kWh/kg, and Gumerman (1978) estimated 16.5 kWh/kg when generating from oxygen. These estimates are consistent with the estimates cited in the energy discussion above. The power costs at 6 cents per kWh are shown in Table 37 using Opatken's estimate for power utilization and contactor efficiencies ranging from 40 to 90%. The 22 kWh/kg O_3

TABLE 37. OZONATION POWER COSTS

PLANT SIZE m^3/d	OZONE DEMAND kg/d	POWER DEMAND kWh/kg O_3	ANNUAL COSTS $	UNIT COSTS $/1,000 m^3
3790	21 - 47	22	10,100 - 22,700	7.32 - 16.39
		30.8	14,160 - 31,700	10.24 - 22.92
37900	209 - 472	22	100,700 - 227,500	7.32 - 16.39
		30.8	141,000 - 318,400	10.19 - 23.02
379000	2092 - 4722	22	1,008,000 - 2,275,000	7.32 - 16.39
		30.8	1,410,000 - 3,185,000	10.19 - 23.02

power demand estimate for oxygen fed ozonators is taken from the energy discussion above because that estimate includes the power cost of producing the oxygen.

Labor costs and supplies for ozone are somewhat less than those for chlorine (Gumerman, 1978). The labor costs per unit of wastewater treated are shown in Table 38. The costs shown are based on Gumerman's hour estimates and an assumed wage rate of \$10/hour.

TABLE 38. OZONATION LABOR COSTS

PLANT SIZE m^3/d	ANNUAL LABOR hrs	ANNUAL COSTS \$	UNIT COSTS \$/1,000 m^3
3790	550	5,500	3.98
37900	900 - 1500	9,000 - 15,000	0.65 - 1.08
379000	4,000 - 7,000	40,000 - 70,000	0.29 - 0.51

Total O & M costs are dependent on the efficiency of ozone absorption and on ozone generation; and, providing that electrical efficiency does not deteriorate, these costs per unit will be nearly invariant with utilization rate. Under the most favorable circumstance, ozone O & M costs are 35 percent higher than chlorine. In the worst case, ozone costs are nine times chlorine costs. As electrical costs increase, ozone economics will deteriorate, unless substantial improvements in ozone generation efficiency can be made. Ozonation O & M costs based on the assumptions stated above are summarized in Table 39.

TABLE 39. OZONATION O & M COSTS SUMMARY \$/1,000 m^3

	PLANT SIZE m^3/d		
	3790	37900	379000
Labor	3.98	0.65 - 1.08	0.29 - 0.51
Power	10.24 - 22.92	7.32 - 16.39	7.32 - 16.39
Supplies	1.95 - 2.82	0.96 - 1.30	0.34 - 0.59
Totals	16.17 - 29.72	8.93 - 18.77	7.95 - 17.49

Ozonation Costs Summary--

Total costs for ozone are the sum of capital and O & M costs and will have a wide range depending on generation efficiency, absorption, and utilization. The ozonation costs per unit of treated wastewater are summarized in Table 40. The values shown are sums of the values shown in Tables 36 and 39 and are constrained by the assumptions described in the discussion above.

TABLE 40. OZONATION COSTS SUMMARY
$\$/1000\ m^3$

PLANT SIZE m^3/d	PERCENT UTILIZATION 60	80	100
3790	41.47 - 75.50	35.15 - 64.06	31.35 - 57.19
37900	21.58 - 39.85	18.42 - 34.58	16.52 - 31.42
379000	13.88 - 26.57	12.40 - 24.30	11.51 - 22.94

Notes - absorbed ozone dosage = 5 mg/L
- minimum unit cost corresponds to 90% transfer efficiency
- maximum unit cost corresponds to 40% transfer efficiency
- interest rate = 9%
- amortization period = 20 years

Ozone costs for disinfection will be significantly higher than chlorine costs. The difference will range between $8.14 and $54.71 per 1000 cubic meters treated. Increases in energy prices and interest rates will both increase the difference between chlorination and ozonation costs. Some perspective can be gained by comparing the difference between ozone and chlorine disinfection with the total cost of wastewater treatment which is assumed for this analysis to be about $430 per 1000 m^3 in a new system. Hence, the choice of ozonation over chlorination will increase total wastewater treatment costs between 2 and 13%.

SECTION 9

RISK MODEL

One objective of this risk analysis project is to develop a risk model from the data base gathered on the risks and benefits associated with the various wastewater disinfectants. The outputs of the risk model serve as the inputs to the decision making process and should be easily interpreted by the decision maker. The model should address, in as quantitative manner as possible, the relative risks and benefits for the disinfectants chlorine, chlorine-dechlorination, ozone, and ultraviolet radiation as well as the no disinfection alternative.

The risk model for this project is based solely on the historical probability of certain events occurring. The health and environment risks associated with the transportation and use of the various disinfectants have been discussed and quantified in the previous sections of the report. This section will present two examples of how this quantitative information can be used by the decision maker in comparing disinfection alternatives.

RISK MODEL DATA BASE

The total reliance on historical data for this risk assessment represents a limitation, and in the case of some disinfectants a severe limitation, to the development of reliable, quantitative information for the decision maker. The risk assessment output can be no better than the quality of the published data and any assumptions required to quantitate a risk. Nevertheless, historical data is often the best source of reliable data, and its sole use does not preclude the development of reliable, quantitative information.

A subjective estimate of the quality of the historical risk assessment data base for the various wastewater disinfectants is presented in Table 41. The ratings are based on both the quantity of published data and their quality. For those data sections classified as insufficient (3) or not applicable (4) a quantitative comparison of risks is not possible.

TABLE 41. ESTIMATE OF HISTORICAL DATA QUALITY

	No Disinf.	Chlorine	Chlorine Dechlor.	Ozone	Ultra-violet
Risks					
On-site Use	4	2	3	3	3
Transportation	4	1	1	4	4
Toxicological effects of residual	4	1	3	3	3
Formation of Hazardous Reaction Products	4	1	3	3	3
Human Health Risks	3	1	3	1	3
Evironmental Risks	3	1	3	3	3
Economic Analysis					
Cost Factors	1	1	1	1	3
Energy Use	4	1	1	1	1

Ratings:

1	sufficient data
2	sufficient data only from parallel industry
3	insufficient data
4	not applicable

The estimate of historical data quality in Table 41 clearly shows the predominance of data on only one disinfection alternative, chlorination. This imbalance in the data base causes chlorination to receive excessive attention in risk assessments drawn from the data base which makes objective assessments difficult to achieve.

RISK MODEL EXAMPLES

Two examples will be presented which illustrate the quantitative aspects of the historical data presented in previous sections. The two examples were chosen to represent maximum and minimum treatment plant capacities with Example A being the small plant. The alternative disinfection processes are limited to chlorination and ozonation in these examples; however, the other alternatives can be incorporated in similar risk analyses in the same manner as demonstrated herein.

The uncertainties associated with the calculations are difficult to quantify but are highly dependent on the assumptions used for such variable factors as applied dosages, size of total workforce, the local ecological community and on the quality of the historical data as discussed above.

Example A

A wastewater treatment plant with a capacity of 3,790 cubic meters per day discharges to a flowing freshwater stream. The total workforce for the treatment facility is 6 full-time employees. The assumed chlorine dosage is 6 mg/L, and the alternative ozone dosage is 5 mg/L with an 85% mass transfer efficiency using air as the feed gas.

Transportation Risks--

Chlorine is to be obtained from a manufacturer located 644 kilometers from the treatment plant. The yearly quantity of chlorine required can be calculated from the applied dosage rate.

$$\begin{aligned}\text{Annual chlorine use} &= 6\ \text{mg/L} \times 3{,}790{,}000\ \text{L/d} \times 365\ \text{days/yr} \\ &\quad \times 10^{-6}\ \text{kg/mg} \\ &= 8300\ \text{kg/yr}\end{aligned}$$

Two examples for transportation are presented to estimate the sensitivity of the transportation mode.

Case 1. Chlorine is transported by truck using 114 kg cylinders the entire 644 kilometers producing an annual haulage of 5,345 metric ton-km. Multiplying this annual haulage by the accident factors contained in Table 9 produces the risk estimates shown below.

deaths - 0
injuries - 0.014/yr
property damage - $1.92/yr
releases - 0.64 kg/yr

Case 2. The chlorine is transported 604 km by railroad then 40 km by truck using 0.91 metric ton containers. This corresponds to 5013 metric ton-km by railroad plus 332 metric ton-km by truck. The risk estimates for this mode of transportation are calculated as above and are shown below.

deaths - 0
injuries - 0.000075/yr
property damage - $0.31/yr
releases - 0.17 kg/yr

The above examples indicate, as does Table 9, that truck transportation of chlorine in 114 kg cylinders has a significantly higher accident rate than other modes of transportation. However, relatively small shipments of chlorine using the small cylinders, as in the above example, do not pose a significant risk for human health and property damage. There is a low sensitivity for transportation mode when dealing with small quantities of chlorine.

On-Site Accidents (Chlorine)--

The on-site accidents information is contained in Tables 2 and 4, and the chlorination risk analysis procedure is described in Section 4. The severity rate for all accidents is calculated as:

Lost work time = 6 employees x 1985 man-hr/employee/yr x 210.9×10^{-6} lost work days/man-hr
= 2.5 lost work days/yr.

The 2.5 lost work day severity rate represents all accident types. To obtain the lost work days from exposure to chlorine the total figure is multiplied by the assumed rate of 4% giving 0.1 lost work days per year from chlorine exposure. This last calculation is based on the assumption that lost work time is about the same for each type of accident.

Energy Use--

Chlorine.-- The total kilowatt-hours required per year is calculated using an efficiency factor of 3.7 kWh per kg Cl_2 from Section 8.

Chlorine energy use = 8300 kg/yr x 3.7 kWh/kg
= 31,000 kWh/yr.

Ozone.--The energy use data for ozone are contained in Section 8 and Table 19. The total ozone required per day is:

Ozone use = 5mg transferred/L x 1 mg applied/0.85 mg transferred x 3,790,000 L/day x 10^{-6} kg/mg
= 22 kg applied/day.

The total kilowatt-hours required per year is calculated using an efficiency factor of 30 kWh/kg ozone. This factor represents an average value since Section 8 data do not clearly indicate any economy of scale.

Ozone Energy Use = 22 kg/day x 365 days/yr x 30 kWh/kg
= 240,000 kWh/yr.

Cost--

The cost data in Section 8 include a variety of assumptions on utilization rates, inflation, interest rates, etc. and the user is directed to the section for details. The calculated costs represent the sum of capital plus operation and maintenance.

Chlorine.--The cost data for chlorine disinfection are summarized in Table 32. With a dosage of 6 mg/L and a utilization of 80%, the total chlorine disinfection cost is calculated as:

$$\text{Chlorine Cost} = \$15.23/1000\ \text{m}^3 \times 3.790\ (1000\ \text{m}^3)/\text{day} \times 365\ \text{days/yr} = \$21{,}000\ /\text{yr.}$$

Ozone.--The cost data for ozone are summarized in Tables 36, 37, 39, and 40. The unit capital costs shown in Table 36 are for 40 and 90% mass transfer efficiency. The unit capital cost for an 85% efficient plant is calculated by interpolation as:

$$\text{Ozone Unit Capital Cost} = \$18.98 + ((34.34 - 18.98) \times ((90-85)/(90-40))) = \$20.52/\ 1000\ \text{m}^3$$

The power cost is calculated from the energy calculation above.

$$\text{Ozone Power Cost} = 240{,}000\ \text{kWh/yr} \times \$0.06\ /\text{kWh} = \$14{,}400\ /\text{yr.}$$

Using unit costs for labor and supplies as shown in Table 39, the ozone costs are calculated as:

$$\text{Ozone Costs} = (\$20.52 + 3.98 + 1.95)/1000\ \text{m}^3 \times 3.79\ (1000\ \text{m}^3)/\text{d} \times 365\ \text{d/yr} + \$14{,}400 = \$51{,}000\ /\text{yr.}$$

Ecological Effects--

The ecological effects of the facility in Example A can be estimated if the following assumptions are accepted.

1. Aquatic organisms do not avoid the effluent, i.e., fish do not swim downstream to lower concentrations.
2. Synergistic effects with other wastewater constituents are not present.
3. The biological community is represented adequately by Bluegill, Channel catfish, Rainbow Trout, Brown Trout, *Daphnia magna*, and an oligochaete worm.
4. Water quality parameters and experimental protocols in the literature are representative of a field situation.
5. Non-lethal concentrations are determined to be one-half the LC-50 concentrations unless species specific data are available (Seegert and Bogardus, 1980).

6. TRC removal mechanisms and dilution in a flowing stream can be estimated by C = Co exp(-kt) where C is stream concentration, Co is initial (diluted) stream concentration at the outfall, t is residence time in the stream, and k is a rate constant.

7. Stream velocity is 3 km/hr, and essentially all TRC is removed or diluted within 8 km from the outfall.

8. Outfall TRC concentration is 0.5 mg/L, which is diluted by the flowing stream at the outfall in a ratio of 1:10.

The summary of literature (Appendix II) on TRC toxicology for the representative aquatic community assumed above is shown in Figure 2, Section 4. The data are very scattered, and as such do not provide a basis for a dose-response function. In particular, the magnitude of a threshold TRC concentration is not consistent thoughout the literature. Sub-lethal effects data are not shown due to their inconsistencies relative to apparently lethal concentrations. Segregation of the data by exposure time or temperature does not significantly decrease variability. However, the assumption of a threshold at 0.5 of LC-50 concentrations provides an approximate method to determine relative population stress on a species basis.

Population stress was determined in a semi-quantitative manner by measuring the graphical distance between the local stream concentration and the bulk of mortality data points in Figure 2. Large graphical distances above local stream concentrations indicated low stress. Small graphical distances above local stream concentrations indicate moderate to high stress. Large distances below local stream concentrations indicate extremely high stress. Equating relative mortality to population stress avoids the misconception of massive aquatic kills. In a chronic situation, populations in high TRC areas will be permanently depressed, and will be comprised of individuals with high resistance to TRC.

Areas of moderate TRC concentrations will have aquatic populations under more moderate stress, including sub-lethal effects such as decreased activity, decreased reproduction, and irritability. However, even in areas of moderate TRC concentrations, weakened individuals will show increased mortality.

Table 42 tabulates values of population stress at various distances downstream from the Example A facility outfall. These values show that trout populations near the outfall will be significantly depressed. Channel catfish and D. Magna populations will have mostly sub-lethal effects near the outfall. Bluegill and Oligochaete worm populations will be unaffected.

Human risk from disinfection of wastewater almost wholly results from potential ingestion. Although it is unreasonable to project direct consumption of water from the outfall of a disinfection facility, it is possible to examine human risk from the proportion of disinfected wastewater which is ingested from public drinking water. Adequate data exist for estimation of human risk from certain carcinogenic products in chlorinated wastewater given that certain bounding assumptions are made.

TABLE 42. EFFECTS OF EXAMPLE A FACILITY ON AQUATIC ORGANISMS

		Species					
Dist. Down-stream km	Conc. mg/l	Blue-gill	Channel Catfish	Rainbow Trout	Brook Trout	D. Magna	Oligo-chate
0	0.050	0	1	3	3	1	0
0.5	0.034	0	0	2	2	1	0
1	0.023	0	0	2	2	1	0
1.5	0.016	0	0	1	1	1	0
2	0.010	0	0	1	0	1	0
3	0.005	0	0	1	0	1	0
4	0.002	0	0	1	0	1	0
6	0.0005	0	0	0	0	0	0

Ratings: 0 = no stress
1 = light stress
2 = moderate stress
3 = high stress
4 = extreme stress

These assumptions are as follows:

1. the risk from consumption of chlorinated wastewater is represented by the risk of ingestion of chloroform and trichloroethylene (other compounds do not have an adequate data base from which to estimate risk),

2. the proportion of chloroform and trichloroethylene to chlorine dose is 0.01 (extrapolated form Naek and Doerr, 1978; Hoehn, et al., 1978),

3. the proportion of chlorinated wastewater entering drinking water systems will not exceed 20%,

4. the human health risk from ingestion of chloroform ranges from 0.37 cancer cases/million people/ug/L (Neal, 1980) to 1.7 cancer cases/million people/ug/L (NAS, 1977),

5. the human health risk from ingestion of trichloroethylene is 0.11 cancer cases/million people/ug/L (NAS, 1977) to 0.13 cancer cases/million people/ug/L (Neal, 1980).

If these assumptions are valid, human risk can be bounded between zero (no wastewater in drinking water) and some value which is an upper bound estimate of human risk.

For the Example A, chlorine dosage of 6 mg/L, chloroform and trichloroethylene concentrations are both 0.06 mg/L (60 ug/L) at the outfall and are diluted to 6 ug/L by the flowing stream.

Using the upper bound cancer risk of 1.7 cancer cases/million people/ug/L and an upper bound percent wastewater in drinking water of 20%, the lifetime risk from chloroform originating from the facility in Example A is:

R(Chloroform) = 1.7 cancer cases/million people/ug/L x 6 ug/L x
0.2 L wastewater/L water consumed
= 2 cancer cases/million people.

Lifetime risk from trichloroethylene originating from the facility in Example A is

R(Trichloroethylene) = 0.13 cancer cases/million people/ug/L x
6 ug/L x 0.2 L wastewater/L water consumed
R = 0.16 cancer cases/million people.
or
R = 0.00000016 cancer cases/lifetime

Thus, total human health risk from wastewater chlorination in the facility of Example A ranges from 0 to 2 x 10^{-7}. It is rather unlikely that 20% of a given drinking water supply will be chlorinated wastewater, since Swayne, et al. (1979) showed that almost half of the population sampled (80 million) ingests drinking water with no chlorinated wastewater, and only 0.7 percent have wastewater concentrations above 5 percent during average flow conditions. However, 2 x 10^{-6} represents an upper bound of risk.

Comparative risk from ozonolysis by-products cannot be estimated due to a lack of data.

Human health risk resulting from recreational use of the stream in Example A can be calculated using the Cabelli model presented in Section 7. Assume k is 0.15 per day and, from the information given above, Qe/(Qe + Qs) is 0.1. Also assume the enterococci concentration of the undisinfected wastewater discharge is 10,000/100 ml. The human health risk from swimming in the receiving stream at a point two days travel distance downstream would then be:

$$\begin{aligned} y &= 11.85 \log(10{,}000 \times 0.1 \times \exp(-0.15 \times 2)) - 0.58 \\ &= 33 \text{ cases of gastrointestinal distress/1000 swimmers.} \end{aligned}$$

Summary--

Table 43 summarizes the risks associated with chlorination and ozonolysis for Example A. Although total disinfection costs and energy use are higher for ozone, human health and environmental risk for ozonolysis are much lower than for chlorination.

TABLE 43. RISKS SUMMARY - EXAMPLE A

Description	Chlorination	Ozonation
Transportation		
Case 1 - truck only		
114 kg cylinders		
Deaths/yr	0	not applicable
Injuries/yr	0.014	
Property damage - \$/yr	\$1.92	
Releases - kg/yr	0.64	
Case 2 - rail + truck		
0.91 metric ton cylinders		
Deaths/yr	0	
Injuries/yr	0.000075	
Property damage - \$/yr	\$0.31	
Releases - kg/yr	0.17	
On-site Accidents - lost work days/yr	0.1	Insuff. Data
Energy Use - kWh/yr	31,000	240,000
Cost - \$/yr	21,000	51,000
Human Health Risk - Cancer cases/lifetime	0 - 2×10^{-6}	Insuff. Data
Ecosystem Effects	trout population stress near the outfall	None

Example B

This wastewater treatment plant with a capacity of 379,000 cubic meters per day is also located on a flowing freshwater stream. The total workforce is 150 full-time employees. The assumed chlorine dosage is 10 mg/L and the absorbed ozone dosage is 5 mg/L with a 90% mass transfer efficiency using oxygen as the feed gas.

Transportation--

Chlorine is to be obtained from a manufacturer located 965 kilometers from the treatment plant. The yearly quantity of chlorine is calculated as follows.

Chlorine Use/yr = 10 mg/L x 379 x 10^6 L/day x 365 days/yr
x 10^{-9} metric tons/mg
= 1,380 metric tons/yr

Annual Haulage = 1,380 metric tons/yr x 965 km
= 1,332,000 metric ton-km/yr

Chlorine for large plants is typically transported by railroad tank cars. The probable risks and damages arising from this chlorine usage are calculated using the factors obtained from Table 9. The resulting estimates are shown below.

Deaths	- 0.0004/yr
Injuries	- 0.02/yr
Property damage	- $79.92/yr
Releases	- 44 kg/yr

On-Site Accidents--

Chlorine.--The on-site accidents data are contained in Tables 2 and 4. The lost work day severity rate for 150 employees is calculated as follows.

lost workdays/yr = 150 employees x 1958 man-hr/employee
x 749.3 x 10^{-6} lost work days/man-hr
x .04 chlorine lost days/lostwork days
= 8.8 lost work days/yr

Energy Use--

Chlorine.--The total kilowatt-hours required per year are calculated using a value of 1.41 kWh/kg Cl_2 as discussed in Section 8.

Chlorine energy use = 1,380,000 kg/yr x 1.41 kWh/kg
= 1,950,000 kWh/yr

Ozone.--The ozone energy utilization rate is assumed to be 22 kWh/kg based on the discussion of oxygen fed ozonators in Section 8.

Ozone production = 5 mg absorbed/L x 1 mg applied/0.9 mg absorbed x 379 x 10^6 L/day
x 365 days/yr x 10^{-9} metric tons/mg
= 768 metric tons/yr

Ozone energy use = 768 metric tons/yr x 22 kWh/kg
x 1000 kg/metric ton
= 16,900,000 kWh/yr

Cost--

Chlorine.--A unit chlorination cost of $4.98/1000 m^3 corresponding to a dosage of 10 mg/L and a utilization rate of 80% was chosed from Table 32 for this example.

Chlorination cost = $4.98/1000 m^3 x 379 (1000 m^3)/day
x 365 days/yr
= $690,000/yr.

Ozone--A unit ozonation cost of $12.40/1000 m^3 corresponding to an absorbed dose of 5 mg/l, a transfer efficiency of 90%, and a utilization rate of 80% was chosen from Table 40 for this example.

Ozonation cost = $12.40/1000 m^3 x 379 (1000 m^3)/day
x 365 days/yr
= $1,720,000/yr

Ecological Effects--

The ecological effects of the facility in Example B can be estimated using the same assumptions discussed for Example A. Example B, however, is assumed to be diluted to a ratio of 1:3 with an undiluted TRC of 1 mg/L at the outfall. These TRC levels account for the much larger outfall volume and chlorination which occurs in a large wastewater facility.

Table 44 shows population stress values for various distances downstream of the Example B facility. All aquatic organisms are stressed near the outfall. Daphnia magna populations may be non-existent near the outfall, which may cause additional stress to predator populations due to lack of food organisms. Channel catfish and Oligochaete worm populations will be highly stressed, and Bluegill populations will be moderately stressed near the outfall.

Human Risk--

The human risk from a facility described as Example B is calculated as in Example A. For Example B with chlorine dosage equal 10 mg/L the chloroform and trichloroethylene concentrations are 100 ug/L. This is diluted to 33 ug/L by the flowing stream at the outfall. Lifetime cancer risks are shown below.

R(Chloroform) = .00000017 cancer cases/ug/L/lifetime
x 33 ug/L x .20 L wastewater/L water

R = 0.000011 cancer cases/lifetime

TABLE 44. EFFECTS OF EXAMPLE B FACILITY ON AQUATIC ORGANISMS

		Species					
Dist. Downstream km	Conc. mg/l	Bluegill	Channel Catfish	Rainbow Trout	Brook Trout	D. Magna	Oligochate
0	0.33	2	3	4	4	4	3
0.5	0.23	2	3	4	4	4	2
1	0.15	1	3	3	3	3	1
1.5	0.10	1	2	3	3	2	1
2	0.07	0	1	3	3	1	0
3	0.03	0	0	2	2	1	0
4	0.01	0	0	1	1	1	0
6	0.003	0	0	0	0	1	0
8	nil	0	0	0	0	0	0

Ratings: 0 = no stress
1 = light stress
2 = moderate stress
3 = high stress
4 = extreme stress

R(Trichloroethylene) = 0.00000013 cancer cases/ug/L/lifetime x 33 ug/L x .20 L wastewater/L water

R = 0.00000086 cancer cases/lifetime

Thus, total human health risk from wastewater chlorination in the facility of Example B ranges from 0 to 0.000011. As in Example A, it is very unlikely that 20% of a given drinking water supply will be chlorinated wastewater (Swayne, et al., 1979), since only 0.7% of the population is exposed to drinking water containing over 5 percent chlorinated wastewater during average flow conditions. Lifetime cancer risk of 0.000011 is an upperbound estimate.

As in Example A, comparative risk for ozonation by-products cannot be estimated due to lack of data.

Human health risk from swimming in the receiving stream for Example B can be calculated using the same assumed values as in Example A except that $Qe/(Qe + Qs)$ is now 0.33. Therefore,

$$y = 11.85 \log(10{,}000 \times 0.33 \times \exp(-0.15 \times 2)) - 0.58$$
$$= 40 \text{ cases of gastrointestinal illness/1000 swimmers.}$$

Summary--

Table 45 summarizes the risks associated with chlorination and ozonalysis for Example B. As was found for Example A, human and environmental risks of ozonalysis is much lower than for chlorination. Energy use and total disinfection costs are still higher for ozonation than for chlorination even though the assumptions selected for Example B extremely favorable to ozonation and somewhat unfavorable to chlorination.

TABLE 45. RISKS SUMMARY - EXAMPLE B

Description	Chlorination	Ozonation
Transportation		
rail tank cars		
Deaths/yr	0.0004	not applicable
Injuries/yr	0.02	
Property damage - $/yr	$79.92	
Releases - kg/yr	44	
On-site Accidents - lost work days/yr	8.8	Insuff. Data
Energy Use - kWh/yr	1,950,000	16,900,000
Cost - $/yr	690,000	1,720,000
Human Health Risk - cancer cases/lifetime	$0 - 12 \times 10^{-6}$	Insuff. Data
Ecosystem Effects	all organisms highly stressed near outfall	None

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APPENDIX A

U.S. CHLORINE PRODUCERS AND PACKAGERS

ALPHABETICAL LISTING OF CHLORINE PRODUCERS IN THE U.S.

M Aluminum Company of America
Point Comfort, Texas (Note 8)

M Amax Specialty Metals Corp.
Rowley, Utah

American Magnesium Co.
Snyder, Texas (Note 8)

M BASF Wyandotte Corp.
Geismar, Louisiana

M Brunswick Chemical Co. Subs.
Brunswick Pulp & Paper Co.
Brunswick, Georgia

Champion International Corp.
1. Canton, North Carolina
2. Houston, Texas

M Convent Chemical Company
1. Calvert City, Kentucky
2. Convent, Louisiana

M Diamond Shamrock Corp.
1. Deer Park, Texas
2. Delaware City, Delaware
3. LaPorte, Texas
4. Mobile, Alabama
5. Muscle Shoals, Alabama

M Dow Chemical USA
1. Freeport, Texas
2. Midland, Michigan
3. Pittsburg, California
4. Plaquemine, Louisiana

M E. I. du Pont de Nemours & Co., Inc.
1. Corpus Christi, Texas
2. Niagara Falls, New York

M Ethyl Corporation
Baton Rouge, Louisiana

M FMC Corporation
South Charleston, West Virginia

M Formosa Plastics Corp., USA
Baton Rouge, Louisiana

Fort Howard Paper Company
1. Green Bay, Wisconsin
2. Muskogee, Oklahoma

M General Electric Co.
Mt. Vernon, Indiana

M Georgia-Pacific Corp.
1. Bellingham, Washington
2. Plaquemine, Louisiana

M Hercules, Inc.
Hopewell, Virginia

M Hooker Chemicals & Plastics Corp.
1. Montague, Michigan
2. Niagara Falls, New York
3. Tacoma, Washington
4. Taft, Louisiana

Hooker - IMC Joint Venture
Niagara Falls, New York

M Int'l Minerals & Chemical Corp.
1. Ashtabula, Ohio
2. Orrington, Maine

M Kaiser Aluminum & Chemical Corp.
Gramercy, Louisiana

M Linden Chemicals & Plastics, Inc.
1. Acme, North Carolina
2. Brunswick, Georgia
3. Linden, New Jersey
4. Moundsville, West Virginia
5. Syracuse, New York

M Mobay Chemical Corp.
Baytown, Texas

M Monsanto Company
Sauget, Illinois

M Olin Corporation
1. Augusta, Georgia
2. Charleston, Tennessee
3. McIntosh, Alabama
4. Niagara Falls, New York

Oregon Metallurgical Corp.
Albany, Oregon

M Pennwalt Corporation
1. Calvert City, Kentucky
2. Portland, Oregon
3. Tacoma, Washington
4. Wyandotte, Michigan

M PPG Industries, Inc.
1. Barberton, Ohio
2. Lake Charles, Louisiana
3. New Martinsville, W. Virginia

M RMI Company[*]
Ashtabula, Ohio

M Shell Chemical Company
Deer Park, Texas

M Stauffer Chemical Company
1. Henderson, Nevada
2. LeMoyne, Alabama
3. St. Gabriel, Louisiana

Titanium Metals Corp. of America[**]
TIMET Divn.
Henderson, Nevada

Vertac Chemical Company
Vicksburg, Mississippi

M Vulcan Materials Company,
Chemicals Division
1. Denver City, Texas
2. Geismar, Louisiana
3. Port Edwards, Wisconsin
4. Wichita, Kansas

M Weyerhaeuser Company
Longview, Washington

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[*] Joint subsidiary of National Distillers & Chemical Corp. and U.S. Steel; sodium & chlorine products sold by U.S. Industrial Chemicals Co., Division of National Distillers.

[**] Titanium Metals Corp. of America is a joint subsidiary of NL Industries, Inc. and Allegheny Ludlum Corp.

U. S. CHLORINE PRODUCERS BY STATES

PLANT LOCATION	COMPANY	C T
Alabama		
Mobile	Thompson-Hayward Chemical Co.	C T
Mobile	Jones Chemicals, Inc.	C T
Muscle Shoals	Thompson-Hayward Chemical Co.	C T
Arizona		
Glendale	McKesson Chemical Co.	C T
Phoenix	Hill Brothers Chemical Co.	C T
California		
Anaheim	Continental Chemical Co.	C T
Milpitas	Jones Chemicals Inc.	C T
Sacramento	Continental Chemical Co.	C T
Santa Fe Springs	McKesson Chemical Co.	C T
Torrance	Jones Chemicals, Inc.	C T
Tracy	All Pure Chemical	C T
Union City	McKesson Chemical Co.	C T
Colorado		
Commerce City	AmeriGas, Inc.	C T
Connecticut		
Waterbury	Hubbard-Hall Chemical Co.	C T
Delaware		
Delaware City	Chloramone Corp.	C T
Florida		
Ft. Lauderdale	Jones Chemicals, Inc.	C T
Jacksonville	Jones Chemicals, Inc.	C T
Leesburg	Allied Universal Corp.	C T
Lloyd	United Chemical Inc.	C T
Miami	Allied Universal Corp.	C T
St. Petersburg	Jones Chemicals, Inc.	C T
St. Petersburg	P B & S Chemical Co., Inc.	C T
Tampa	Thompson-Hayward Chemical Co.	C T
Tampa	Moreland-McKesson Chemical Co.	C T
Georgia		
Powder Springs	Thompson-Hayward Chemical Co.	C T
Savannah	Ashland Chemical Co.	C T
Hawaii		
Honolulu	Brewer Chemical Corp.	C -
Illinois		
Lemont	Alexander Chemical Corp.	C T
Lemont	K.A. Steel Chemicals Inc.	- T
Indiana		
Beech Grove	Jones Chemicals, Inc.	C T
LaPorte	Midwest Chlorine Corp.	C T
Terre Haute	Ulrich Chemical Inc.	C T
Iowa		
Burlington	McKesson Chemical Co.	C -
Kansas		
Iola	Treat-Rite Water Labs., Inc.	C -
Wichita	Vulcan Materials Co. (Notes P.2)	- T
Kentucky		
Henderson	P B & S Chemical Co., Inc.	C T
Louisiana		
Geismar	McKesson Chemical Co.	C T
Lake Charles	Thompson-Hayward Chemical Co.	C T
Napoleonville	Bartlett Chemicals, Inc.	C T
Reserve	Jones Chemicals, Inc.	C T
St. Gabriel	Thompson-Hayward Chemical Co.	C T
Maryland		
Baltimore	Delta Chemical Mfg. Co.	C T
Michigan		
Wyandotte	Jones Chemicals, Inc.	C T
Minnesota		
Minneapolis	Hawkins Chemical Co. Val-Tex Div.	C T
St. Paul	Van Waters & Rogers Divn.	C T
Mississippi		
Vicksburg	Vertac Chemical Co. (Note P.4)	C T
Missouri		
Festus	Jones Chemicals, Inc.	C T
St. Louis	McKesson Chemical Co.	C T
Montana		
Billings	AmeriGas, Inc.	C T
Nebraska		
Omaha	Van Waters & Rogers Divn.	C T
Nevada		
Henderson	Jones Chemicals, Inc.	C T
Reno	Sierra Chemial Co.	C T
New Hampshire		
Merrimack	Jones Chemicals, Inc.	C T
Merrimack	New England Chemical Co.	C T
New Jersey		
South Kearny	Allied Universal Corp.	C T
South Kearny	Kuehne Chemical Co.	C T
New Mexico		
Albuquerque	AmeriGas Inc.	C T
New York		
Caledonia	Jones Chemicals, Inc.	C T
Rensselaer	Ashland Chemical Co.	C T
Utica	Monarch Chemical Co.	C T
Warwick	Jones Chemicals, Inc.	C T
Watkins Glen	Ryeler, Inc.	C T
North Carolina		
Charlotte	Jones Chemicals, Inc.	C T
Greensboro	Moreland-McKesson Chemical Co.	C T
Thomasville	Thompson-Hayward Chemical Co.	C T
Ohio		
Barberton	Jones Chemicals, Inc.	C T
Cincinnati	Herbert Verkamp Cal. Chem. Co.	C T
Oklahoma		
Oklahoma City	Ashland Chemical Co.	C T
Sand Springs	Kimball Chemical Co., Inc.	C T
Oregon		
Portland	Pennwalt Corporation (Note P)	C T
Pennsylvania		
Bunola	United Chemical Inc.	C T
Middletown	United Chemical Inc.	C T
South Carolina		
Chapin	United Chemical Inc.	C T
Spartanburg	Moreland-McKesson Chemical Co.	C T
Tennessee		
Chattanooga	Mayo Chemical Co.	C T
Chattanooga	Moreland McKesson Chemical Co.	C T
Chattanooga	P B & S Chemical Co.	C T
Texas		
Abilene	Carroll Chem. & Cryogenics Inc.	C T
Corpus Christi	AmeriGas, Inc.	C T
Dallas	Thompson-Hayward Chemical Co.	C T
El Paso	AmeriGas, Inc.	C T
Fort Worth	AmeriGas, Inc.	C T
Harlington	South Texas Chlorine, Inc.	C T
Houston	J.W. Jones Chemical Co.	C T
LaPorte	Dixie Chemical Co.	C T
Snyder	AmeriGas, Inc.	C T
Utah		
Salt Lake City	Thatcher Chemical Co.	C T
Salt Lake City	Wasatch Chemical Co.	C T
Virginia		
Milford	Jones Chemicals, Inc.	C T
Petersburg	Dominion Chemical Co.	C -
Suffolk	United Chemical Inc.	C T
Washington		
Tacoma	Jones Chemicals, Inc.	C T
Tacoma	Pennwalt Corporation (Note P)	C T
West Virginia		
New Martinsville	P B & S Chemical Co., Inc.	C T
St. Albans	P B & S Chemical Co., Inc.	C T
Wisconsin		
Hudson	Jones Chemicals, Inc.	C T
Oshkosh	Hydrite Chemical Co.	C T

(t = ton containers; c = cylinders)

APPENDIX B

SUMMARY OF REPORTED CHLORINE EFFECTS ON FRESHWATER ORGANISMS

Species	Endpoint	Exposure Time Min.	Temp. of Test Solution	Conc.	Reference
(Plants)					
Chlorophyta	DG-50	1,440	--	(FRC,CRC) 0.18	Kott, et al., 1966
Algae	DG-50	300	--	(FRC,CRCO 0.4	Kott.and Edlis, 1969
Pyrenoidosa					
Chlorella var.	DG	4,320/	--	(Ca hypo.) 2.0	Palmer and Maloney, 1955
Ob. Scenedesmus	DG	4,320/	--	(Ca hypo.) 2.0	Palmer and Maloney, 1955
Scenedesmus Sp.	MT	5,760	--	(CRC) 10.0	Bringham and Kuhn, 1959
Chrysophyta					
Gomphonema Parv.	DG	4,320/	--	2	Palmer and Maloney, 1955
Palea Nitzschia	DG	4,320/	--	2	Palmer and Maloney, 1955
Cyanophyta					
Cylindrospermum	DG	4,320/	--	2	Palmer and Maloney, 1955
Aeru. Microcystis	DG	4,320/	--	2	Palmer and Maloney, 1955
Miscell-Phytoplants	Stops Growth	NG	--	0.4	Brook and Baker,(1972)
(Invertebrates)					
Water louse	No Repro	60	--	(TRO) 0.5	Holland, G.J., 1956
Protozoa	Some Mortality	1/min	--	(FRL) 2-8	Ranganathan and Small, 1970
Cyclops. Sp.	Some Mortality	30	--	(TRC) 1.0	Adams, B.A., (1927)

Species	Endpoint	Exposure Time Min.	Temp. of Test Solution	Conc.	Reference
Water Flea Daphnia magna	MT	2,880	--	(CRC) 4.0	Bringman, G., and Kuhn, 1959
Water Flea Daphnia magna	LC-1--	240	--	0.125	Brungs, W.A., 1973
Water Flea Daphnia magna	D Repro.	20,160	--	(TRC) 0.002	Arthur, J.W., et al. 1974
Water Flea Daphnia magna	LC-100	4,320	--	(TRC) 0.5	Ellis, M.M., (1937)
Water Flea Daphnia magna	Some Mortality	60	--	(TRC) 0.5	Adams, B.A., (1927)

MT = Mortality Threshold
DG = Decreased Growth
NE = No Effect
DP - (Decreased Photosynthesis)

NG = Not Given
TRC= Total Residual Chlorine
FRC= Free Residual Chlorine
MCA = Monochloramine
CRC = MCA + di and tri

Species	Endpt.	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Invertebrates						
Scud	LC-50	2,880	15°	(TRC)/ 0.023	FT	Gregg, B.C., 1974 and
Scud	LC-80	151,200	--	(CRC) 0.035	FT	Arthur, Eaton, 1971 and
Scud	LC-50	5,760	--	(CRC) 0.22	FT	Arthur, Eaton, 1971 and Arthur, Eaton, 1975
Scud	0% Repo	151,200	--	(CRC) .0034	FT	
Scud	D Survival	161,280	--	(TRC) .054	FT	Arthur, J.W., et al. 1974
Scud	D Repo	201,600	--	(TRC) 0.019	FT	Arthur, J.W., et al. 1974
Scud	NE	43,200	--	(TRC) 0.135	FT	Arthur, J.W., et al. 1974
S	LC-50	1,440	--	(TRC) 0.900	FT	Arthur, J.W., et al. 1974
Orlonectes Virilis Crayfish	LC-50	10,080	--	(TRC) 0.780	FT	Arthur, J.W., et al. 1974
Arthropoda-Insecta Mayfly	LC-50	480	25°	0.502	FT	Gregg, B.C., 1974
Centroptium, Sp. Mayfly	LC-50	1,440	6°	(TRC) 0.071	FT	Gregg, B.C., 1974
Chironomus Sp. Midge larvae	LC-80	1,440	--	(TRC) 7.0	S	Buchman, W., 1933

Species	Endpt.	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Ephemerella lata Mayfly	LC-50	2,880	15°	(TRC) 0.027	FT	Gregg, B.C., 1974
Iron humeralis Mayfly	LC-50	480	15°	(TRC) 0.046	FT	Gregg, B.C., 1974
Isonychia Sp. Mayfly	LC -50	2,880	6°	0.0093	FT	Gregg, B.C., 1974
Hydropsyche bifida Caddisfly	LC-50	480	25°	0.396	FT	Gregg, B.C., 1974
Caddisfly	LC-50	10,080	--	0.55	FT	Gregg, B.C., (1974)
Peltoperia maria Stonefly	LC-50	2,880	15°	0.020	FT	Gregg, B.C., 1974
Annelida						
Oligochaete worm	LC-95	35	--	(FRC) 1.0	FT & S	Learner and Edwards 1963
Oligochaete worm	LC-95	34	--	(TRL) 1.0	S	Collins, J.S., 1958
Oligochaete worm	Disintegration	30	--	(FRC) 0.5	S	Hart, K.M., 1957
Nematode worm	LC-50	30	--	(FRC) 91.0	S	Chang et al. 1960
Nematode worm	LC-50	120	--	13.0	S	Chang et al. 1960
Nematode worm	LC-100	150	--	(TRC) 20.0	S	Collins, J.S., 1958
Nematode worm (Immature)	LC-100	90	--	3.0	S	Collins, J.S., 1958

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Mollusca						
Operculate snail	LC-50	20,160	--	(TRC) > 0.810	FT	Arthur, J.W., et al. 1974
Operculate snail	LC-50	5,760	25°	(TRC) 0.044	FT	Gregg, B.C., 1974
(Ancalosa) "	LC-50	5,760	25°	(TRC) 0.086	FT	Gregg, B.C., 1974
Pulmonate snail	LC-50	20,160	—	(TRC) > 0.810	FT	Arthur, J.W., et al. 1974
P. heterostropha "	LC-50	5,760	25°	(TRC) 0.258	FT	Gregg, B.C., 1974
Vertebrate Animals						
Tadpole	LC-100	510	--	2.4	S	Painkkar, B.M., 1960
Fish						
Gizzard Shad	MT	10	--	0.62	S	Truchan, J.G., and Basch, R.E., 1971
Coho salmon	MT	1,440	--	0.016	—	Rosenberger, D.R., 1972
Coho salmon	MT	5,760	--	0.004	—	" " "
Coho salmon	—	—	--	—	—	—
Coho salmon	"	"	6°	0.68	--	Heath, A.G., 1978
Coho salmon	"	"	12°	0.64	--	Heath, A.G., 1978
Coho salmon	"	8,640	12°	0.55	—	Heath, A.G., 1978
Coho salmon	"	"	6°	0.54	--	Heath, A.G., 1978
Coho salmon	"	5,760	—	0.89	FT	Ward, R.W., and DeGrave, G.M., (1978)
Coho salmon	"	"	6°	0.677	FT	Heath, A.G., (1977)
Coho salmon	"	4,320	6°	0.181	FT	Heath, A.G., 1977
Coho salmon	"	5,760	10-15°	(CRC) 0.060	FT	Larson, G.L., et al. 1977
Coho salmon	"	"	12°	0.289	S	Marking, L.L., and Bills, T.D., 1977
Coho salmon (Alevin)	"	"	9.9-10.2°	(TRC) .080-0.083	FT	Larson, G.L., et al. 1978
Coho salmon	"	30	10.3°	0.56	S	Seegert, G.L., and Brooks, A.S., 1978
Coho salmon (Fry)	"	30	10.3°	(TRC) 0.079	S	Larsdn, G.L., et al. 1978
Coho salmon (Juvenile)	"	30	10.5-15.0°	(TRC) 0.053-0.082	S	Larson, G.L., et al. 1978
Coho salmon	"	30	20.1°	0.29	S	Seegert, G.L., and Brooks, A.S., 1978

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/1	Test Type	Reference
Brown Trout	MT	1,440	—	(FRC) 0.35	S	Rushton, W., 1921
"	LC-50	120	--	0.50	S/CF	Pike, D.J., 1971
"	LC-50	180	--	0.09	S/CF	" " "
"	LC-50	360	--	0.05	S/CF	" " "
"	LC-50	660	--	0.02	S/CF	" " "
(Fingerlings) "	LC-50	90	--	0.5	S	Pyle, E.A., 1960
Brook Trout	MT	10,080	--	(TRC) 0.01	FT	Dandy, J.W.T., 1972
Brook Trout	Activity Depressed	10,080	--	(TRC) 0.005	FT	Dandy, J.W.T., 1971
" "	MT	10,080	--	(CRL) 0.01	S	Coventry, F.L., et al 1935
" "	LC-100	2,880	--	(CRC) 0.05	S	Coventry, F.L., et al 1935
" "	LC-50	5,760	--	(TRC) 0.06	FT	Wolf, E.G., et al. 1976
" "	LC-100	1,440	--	(TRC) 10.0	S	Belding, D.L., 1927
"	LC-50	720	—	(TRC) 0.360	FT	Authur, J.W., et al. (1974)
" "	LC-50	5,760	20°	(TRC) 0.012	FT	Thatcher, T.O., et al 1976
" "(Alevin)	LC-50	5,760	10.6- 10.8°	(TRC) 0.09-.1	FT	Larson, G.L., et al. 1978
" "(Fry)	LC-50	5,760	10.8°	(TRC) 0.08/8	FT	Larson, G.L., et al. 1978
" "(Juvenile)	LC-50	5,760	11.1- 11.3°	(TRC) 0.088-.09	FT	Larson, G.L., et al. 1978
Northern Pike	LC-100	1,800	4.5-7°	(FRC) 0.70	S/FT	Ebeling, G., 1931
Grass Pickerel	LC-100	60	--	(TRC) 1.0	S	Hubbs, C.L., 1930
White Sucker	LC-100	60	--	(TRC) 1.0	S	Fobes, R.L., 1971
" "	LC-50	720	--	(TRC) 0.248	FT	Authur, J.W., et al. 1974
" "	LC-50	5,760	12°	0.379	S	Marking, L.L., and Bills, T.D., 1977
Goldfish	LC-100	240	--	(TRC)	S	Panikkar, B.M., 1960
"	LC-100	1,440	--	(FRC) 0.3	FT	McCauley, R.W., and Scott, D.P., 1060

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Goldfish	MT	480	--	(TRC) 1.0	FT	Zimmerman, P.W., and Berg, R.O., 1934
Goldfish	LC-50	1,440	20-22.5°	0.49	FT	Tsai, C., and
Goldfish	LC-50	2,880	20-22.5°	(TRC) 0.38	FT	McKee, J.A., 1978
Goldfish	LC-50	4.320	20-22.5°	(TRC) 0.35	FT	Tsai, C., and
Goldfish	LC-50	5,760	20-22.5°	(TRC) 0.35	FT	McKee, J.A., 1978
Goldfish	LC-50	5,760	12°	(TRC) 1.18	S	Marking, L.L., and Bills, T.P., 1977
Goldfish	LC-50	5,760	25°	0.153-0.21	FT	Ward, R.W., and DeGrave, G.M., 1978
Carp	LC-80	6,000	--	(FRC) 0.7	S/FT	Ebeling, G., 1931
"	MT	65	--	(TRC) 0.72		Brook, A.J., and Baker, A.L., 1972
"	LC-50	5,760	12°	(TRC) 0.80	S	Marking, L.L., and Bills, T.D., 1977
"	LC-50	5,760	6°	(CRC) 1.72	FT	Heath, A.G., 1977
"	LC-50	5,760	6°	(FRC) 0.538	FT	Heath, A.G., 1977
"	LC-50	80	10°	(CRC) 2.37	-	Brooks, A.S., and Seegert, G.L., 1978
"	LC-50	2,880	20°	(CRC) 1.82	-	Brooks, A.S., and Seegert, G.L., 1978
"	LC-50	2,880	30°	(CRC) 1.50	-	Brooks, A.S., and Seegert, G.L., 1978
Golden Shiner	Death	0.17	--	(FRC) 73,000	S	Lewis, W.M., and Ulrich, M.G., 1967
Golden Shiner	LC-100	240	--	(TRC) 0.80	S	Panikkar, B.M., 1960
Golden Shiner	LC-50	5,760	6°	(FRC) 0.269	FT	Heath, A.G., 1977
Golden Shiner	LC-50	5,760	24°	(FRC) 0.193	FT	Heath, A.G., 1977
Golden Shiner	LC-50	5,760	6°	(CRC) 0.724	FT	Heath, A.G., 1977

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Golden Shiner	LC-50	5,760	24°	(CRC) 0.930	FT	Heath, A.G., 1977
Golden Shiner	LC-50	5,760	25°	(TRC) 0.04	FT	Heath, A.G., 1977
Golden Shiner	LC-50	1,800	5°	(TRC) 0.84	-	Heath, A.G., 1978
Golden Shiner	LC-50	810	24°	(TRC) 0.26	-	Heath, A.G., 1978
Golden Shiner	LC-50	8,640	5°	0.18	-	Heath, A.G., 1978
Golden Shiner	LC-50	8,640	24°	0.18	-	" " "
" "	"	2,880	5°	(MCA) 0.99	-	" " "
" "	"	2,880	24°	(MCA) 1.09	-	" " "
" "	"	8,640	5°	(MCA) 0.64	-	" " "
" "	"	8,640	24°	(MCA) 0.92	-	" " "
" "	Avoidance	10	12°	(TRC) 0.209	FT	Larrick, S.R., and Cherry, D.S., and Dickson, K.L., and Cairns, J., 1973
" "	"	"	18°	(TRC) 0.199	FT	
" "	"	"	24°	(TRC) 0.395	FT	
" "	"	"	30°	(TRC) 0.203	FT	" " "
" "	"	"	12°	(CRC) 0.086	FT	" " "
" "	"	"	18°	(CRC) 0.112	FT	" " "
" "	"	"	24°	(CRC) 0.255	FT	" " "
" "	"	"	30°	(CRC) 0.114	FT	" " "
" "	"	"	12°	(FRC) 0.123	FT	" " "
" "	"	"	18°	(FRC) 0.086	FT	" " "
" "	"	"	24°	(FRC) 0.139	FT	" " "
" "	"	"	30°	(FRC) 0.088	FT	" " "

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Golden Shiner	Avoidance	10	12°	(HOCl) 0.044	FT	" " "
" "	"	"	18°	(HOCl) 0.027	FT	Larrick, S.R., and Cherry, D.S., and Dickson, K.L., and Cains, J., 1978
" "	"	"	24°	(HOCl) 0.045	FT	
" "	"	"	30°	(HOCl) 0.033	FT	
" "	"	12-30	--	(FRC) .015- .017	FT FT	" " " " " "
Common Shiner	LC-100	76	--	(TRC) 0.7	S	Hubbs, I.L., (1930)
" "	LC-50	5,760	25°	0.51	FT	Ward, R.N., and Degrave, G.M., 1978
" "	LC-50	2,880	10°	(TRC) 0.78	FT	Brooks, A.S., and Seegert, G.L., 1978
" "	LC-50	2,880	20°	(TRC) 0.59	FT	" " "
" "	LC-50	2,880	30°	(TRC) 0.45	FT	" " "
Roseyface Shiner	LC-100	180	--	(TRC) 0.07	FT	Hubbs, C.L., 1930
" "	LC-100	79	--	(TRC) 0.07	FT	" " "
Spottail Shiner	LC-50	30	10.1 - 20°	(TRC) 2.41-0.53	S	Seegert, G.L., and Brooks, A.S., 1978
Pugnose Shiner	LC-50	5,760	25°	(TRC) 0.045	FT	Ward, R.W., and DeGrave, G.M., 1978
Spotfin Shiner	LC-50	2,880	10°	(TRC) 0.65	FT	Brooks, A.S., and Seegert, G.L., 1978
			20°	0.59	FT	" " "
			30°	0.41	"	
Emerald Shiner	LC-50	2,880	10°	(TRC) 0.63	FT	Brooks, A.S., and Seegert, G.L., 1978
" "	LC-50	2,880	20°	(TRC) 0.51	FT	" " "
" "	LC-50	2,880	30°	(TRC) 0.35	FT	" " "
" "	LC-50	30	10°	-- 0.95	FT	Fandrei, G.L., 1977
" "	LC-50	30	25°	-- 0.28	FT	Fandrei, G.L., 1977

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc. mg/L	Test Type	Reference
Blunt Nose Minnow	LC-100	61	--	(TRC) 0.7	FT	Hubbs, C.L., 1930
(larvae) Fathead Minnow	LC-60	43,200	--	(CRC) 0.108	FT	Arthur, J.W., and Eaton, J.G., 1971
" (larvae)	DG-68	43,200	--	(CRC) 0.108	FT	" " "
Fathead Minnow	5^{10}- D. Repro.	10,080	--	(CRC) 0.043	FT	" " "
"	LC-50	5,760	--	(TRC) 0.08-0.19	FT	Zillich, J.A., 1969
"	MT	5,760	--	(TRC) 0.05	FT	" " "
"	LC-50	7,200	--	(TRC) 0.02	FT	Basch, R.E., et al. 1971
"	LC-50	720	--	(TRC) 0.185	FT	Authur. J.W., et al. 1974
"	D. Repro.	100,800	--	(TRC) 0.110	FT	" " "
"	LC-50	5,760	25°	(TRC) 0.082-.095	FT	Ward, R.N., and DeGrave, G.M., 1978
"	LC-50	5,760	12°	(TRC) 0.998	S	Marking, L.L., and Bills, T.D., 1977
"	D. Repro.	--	--	(CRC) 0.085	FT	Zillich, J.A., 1969
"	No Spawning	--	--	(CRC) 0.043	FT	" " "
"	LC-100	--	--	(CRC) 0.16-0.21	FT	" " "
"	LC-100	4,320	--	(CRC) 0.154	FT	Arthur, J.W., and Eaton, J.G., 1971
"	Reduced Offspring	10,080	--	(CRC) 0.43	FT	" " "
Pimephales Vigilar	Avoidance	--	--	0.50	--	Bogardus, R.B., et al 1978
Minnow	LC-100	79	--	(TRC) 0.7	FT	Hubbs, C.L., 1930
Rudd	LC-100	2,460	--	(FRC) 0.7	FT	Ebeling, G., 1931

Species	**Endpoint**	**(Min.) Exposure Time**	**Temp. °C**	**(Type) Conc. mg/L**	**Test Type**	**Reference**
Tench	LC-20	6,000	--	(FRC) 0.7	FT	Ebeling, G., 1931
Black Bullhead	LC-50	1,440	--	(FRC) ~4.5	S	Panikkar, B.M., 1960
Black Bullhead	MT	25	--	(TRL) 1.36	FT	Truchan, J.G., and Basch, R.E., 1971
" "	LC-50	5,760	12°	(TRC) 1.41	FT	Marking, L.L., and Bills, T.D., 1977
Channel Catfish	LC-50	5,760	6°	(FRC) 0.082	FT	Heath, A.G., 1977
" "	LC-50	5,760	24°	(FRC) 0.064	FT	" " "
" "	LC-50	5,760	12°	(TRC) 0.156	S	Marking, L.L., and
" "	LC-50	2,880	5°	(FRC) 0.20	FT	Heath, A.G., (1978)
" "	LC-50	2,880	24°	(FRC) 0.14	FT	" " "
" "	LC-50	7,200	5°	(FRC) 0.05	FT	" " "
" "	LC-50	7,200	24°	(FRC) 0.05	FT	" " "
" "	LC-50	5,760	6°	0.28	FT	" " "
" "	LC-50	5,760	24°	0.33	FT	" " "
" "	LC-50	8,640	6°	0.21	FT	" " "
" "	LC-50	8,640	24°	0.25	FT	" " "
Small Mouth Bass	LC-50	900	--	(FRC) 0.5	S	Pyle, E.A., 1960
Large Mouth Bass	LC-50	5,760	25°	(TRC) 0.241	FT	Ward, R.W., and DeGrave, G.M., 1978
" "	LC-50	1,440	--	(TRC) 0.494	FT	Authur, J.W., 1974
Striped Bass	LC-50	1,440	--	0.3	S	Hughes, J.S., 1970
" "	LC-50	2,880	--	- 0.25	S	" " "
White Bass	LC-50	60	--	0.25-.035	FT	Brungs, W.A., (1973)
" "	Avoidance	45	--	0.035	FT	Grieve, J.A., et al. 1978
Green Sunfish	LC-50	1,440	--	(FRC) 2.0	S	Panikkar, B.M., 1960

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc.	Test Type	Reference
Green Sunfish	LC-100	--	--	(CRC) 0.4	S	Coventry, F.L., et al 1935
Sunfish	LC-50	5,760	25°	(TRC) 0.195-.278	FT	Ward, R.W., and DeGrave, G.M., 1978
Green Sunfish	LC-50	5,760	12°	(TRC) 1.28	S	Marking, L.L., and Bills, T.D., 1977
Crapple	LC-50	5,760	25°	(TRC) 0.127	FT	Ward, R.W., and DeGrave, G.M., 1978
Mosquito Fish	MT	4,320	--	(FRC) 0.5-1.0	--	Gromou, A.S., 1944
Eel	MT	6,000	--	(TRC) 0.7	FT	Ebeling, G., 1931
Black Crapple	MT	25	--	(TRC) 1.36	FT	Truchan, J.G., and Basch, R.E., 1971
Yellow Perch	MT	65	--	0.72	FT	" " "
Yellow Perch	LC-50	720	--	(TRC)	FT	Authur, J.W., et al. 1974
ellow Perch	LC-50	5,760	12°	(TRC) 0.558	S	Marking, L.L., and Bills, T.D., 1977
Walleye	LC-50	720	--	(TRC) 0.267	FT	Authur, J.W., et al. 1974
"	LC-50	5,760	25°	(TRC) 0.108	FT	Ward, R.W., and DeGrave, G.M., 1978
Minnows "Killies"	NE	120	--	(FRC) 0.3	S	Schaut, G.C., 1939
Notropis Volulellus	Avoidance	--	--	0.005	—	Bogardus, R.B., et al. 1978
" Belnnis	"	—	--	0.150	--	" " "
Alewife	LC-50	30	10.5°	(TRC) 2.15	S	Seegert, G.L., and Brooks, A.S., 1978
"	LC-50	30	15.2°	(TRC) 2.27	S	" " "
"	LC-50	30	20.1°	1.70	S	" " "
"	LC-50	30	25.1°	0.96	S	" " "
"	LC-50	30	29.8°	0.30	S	
White Sucker	LC-50	5,760	12°	0.379	S	Marking, L.L., and Bills, T.D., 1977
Cutthroat Trout	LC-50	5,760	10.1- 15.1°	(TRC) 74.5-94.7	FT	Larson, G.L., et al. 1978
" (Juvenile)						
Lake Trout	"	"	12°	(TRC) 0.200	S	Marking, L.L., and Bills, T.D., 1977

Species	Endpoint	(Min.) Exposure Time	Temp. °C	(Type) Conc.	Test Type	Reference
Lake Trout	LC-50	5,760	14°	(TRC) 0.060	FT	Ward, R.W., and DeGrave, G.M., 1978
Bluegill	LC-50	5,760	15-32°	(TRC) 0.44	FT	Bass, M.L., et al. 1977
"	LC-50	5,760	6-32°	(TRC) 0.43-0.47	FT	Bass, M.L., and Heath, A.G., 1977
"	LC-50	5,760	12°	(TRC) 0.555	S	Marking, L.L., and Bills, T.D., 1977
"	LC-50	2,880	10°	(CRC) 3.00	FT	Brooks, A.S., and Seegert, G.L., 1978
"	LC-50	2,880	20°	(CRC) 1.72	FT	" " "
"	LC-50	2,880	30°	(CRC) 1.23	FT	" " "
"	LC-50	2,880	25°	(TRC) 0.54	FT	Heath, A.G., 1978
"	LC-50		32°	(TRC) 0.47	FT	" " "
"	LC-50	10,080	6°	(TRC) 0.33	FT	" " "
"	LC-50		25°	(TRC) 0.37	FT	" " "
Blacknose Dance	Avoidance	--	--	(FRC) 0.07.0.21.0.47	--	Fava, Tsai 1978
"	"	--	--	(MCA) 0.07 and 0.17.	--	" " "

APPENDIX C

SUMMARY OF TOXIC EFFECTS OF CHLORINE TO MARINE AQUATIC LIFE

ˀpecies	Endpoint	(Min.) Exposure Time	Test Temp.	(Type) Conc. mg/L	Test Type	Reference
Plants	DG-71%	240	--	- 0.1	--	Carpenter, E.J., et al. 1972
Phytoplankton	DP-50%	--	--	- 0.03	--	Morgan, R.P. and Stross, R.G., 1969
Chlorophyta (Algea)	DG	5-10	--	- 1.5	--	Hirayama, K., and Hirano, R., 1970
Blue-green "	DG-50	1,440	--	- 0.11	--	Gentile, J.H., et al. 1974
Yellow-green Algae						
Asterionella japonia	DG-50	0.27	--	0.4	--	Gentile, J.H., 1972
. " "	DG-50	2	--	0.2	--	" " "
Chaetoceros decipiens	DG-50	1,440	--	0.14	--	Gentile, J.H., et al. 1974
" didymum	DG-50	1,440	--	0.125	--	" " "
Detonula Confervacea		0.6	--	0.8	--	Gentile,J.H., 1972
Skeletonema costatu	DG-50	1,440	--	0.095	--	Gentile, J.H., et al. 1974
" "	DG-50	1.70	--	0.6	--	Gentile, J.H., 1972
halassiosira Norden	DG-50	1,440	--	0.195	--	Gentile, J.H. et al. 1974
" Pseudonona	DG-50	1,440	--	0.077	--	Morgan, R.P. and Stross, R.G., 1969
" "	DG-50	6.8	--	0.2	--	" " "
" "	DG-50	0.3	--	0.5	--	Gentile, J.H., 1972
" Rotula	DG-50	1,440	--	0.33	--	Gentile, J.H., et al. 1974
Monochrysis lutheri	DG-50	1,440	--	0.2	--	" " "
Rhodomonas ballica	DG-50	1,440	--	0.11	--	" " "
Phyto plankton	DP-95%	10	--	1.0	--	Davis, M.H., and Coughlan, J., 1978
Giant Kelp	DP-50%	5,760	--	5-10	--	Clendenning, K.A., and North, W.J., 1959
Invertebrate Animals						
Sea anemone	NE	21,600	--	1.0	--	Turner, H.J., et al. 1948
Hydroid	DG	180	--	2.5	--	McLean, R.I., 1972
Polychaete worm	17% Decreased Sperm mortality	5	--	0.2	--	Muchmore, D., and Epel, P., 1973
"	70% Decreased Sperm mortality	5	--	0.4	--	" " "

Species	Endpoint	(Min.) Exposure Time	Test Temp.	(Type) Conc. mg/L	Test Type	Reference
Crassostnea vir. Oyster	46% Decreased Cillary	--	--	0̄.2	--	Galtsoff, P.S., 1946
Crassostnea vir. Oyster	Pumping Threshold	20-90	--	1̄.0	--	Galstoff, J.H., et al. 1946
" "	50% Decrease In Time open	4,320	--	0̄.18	--	Patrick, R., and McLean, R., 1970
Ostrea edul. Oyster Larvae	Swimming Stopped	2	--	0̄.5	--	Waugh, C.D., 1964
Ostrea edul. Oyster	NE	10	30°	2̄.5	--	Hydro. Bio Study 1967
Mytilus edulis (Mussel)	LC-100	21,600	--	1̄.0	--	Turner, H.J., et al. 1948
Mytilus edulis (Mussel)	LC-100	7,200	--	2̄.5	--	" " "
Mytilus edulis (Mussel)	Unattached Young	--	--	0̄.02-0.05	--	James, W.C., 1967
Gastro pods	Stop Growth	--	--	0̄.2	--	White, W.R., 1966
Aeartia tonsa (Copepod)	LC-30	2	20°	0̄.75	--	Dressel, D.M., 1971
Aeartia tonsa (Copepod)	LC-70	2	25°	0̄.75	--	" " "
" "	LC-50	120	--	1̄.0	--	Gentile, J.H., et al. 1974
" "	LC-50	0.7	--	1̄0.0	-	" " "
" "	LC-90	5	--	2̄.5	--	McLean, R.I., 1976
Eurytemora aff. (Copepod)	LC-50	360	--	1̄.0	--	Gentile, J.H., et al. 1974
Eurvtemora aff. (Copepod)	LC-50	2	--	10̄.0	--	" " "
Pseudodiaptomus coro. (Copepod)	LC-50	45	--	2̄.5	--	" " "
Pseudodiaptomus coro. (Copepod)	LC-50	5	--	10̄.0	--	" " "
Adult copepod	LC-87	2,880	--	71̄.0	--	Davis, M.H.
" "	LC-69	2,880	--	0̄.8-1,0	--	Coughlan, J., 1978
" "	LC-22	2,880	--	0̄-0.25	--	" " "
Copepod Stages	LC-70	2,880	--	0̄.8-1.0	--	" " "
" "	LC-77	2,880	--	1̄.0	--	" " "
" "	LC-26	2,880	--	0̄-0.25	--	" " "
ɔmarus ammer. (Decopepod)	LC-50	5,760	--	(CRC) 0.05	--	Cappuzzo, J.M., et al. 1976

Species	Endpoint	(Min.) Exposure Time	Test Temp.	(Type) Conc. mg/L	Test Types	Reference
Palaemonetis pegis (Decopepod)	LC-50	5,760	—	(TRC) 0.22	--	Bellanca, M.A..and Bailey, D.S., 1977
Barnacle larvae	LC-80	5	--	$\bar{2}.5$	—	McLean, R.I., 1973
Barnacles	LC-90-100	21,600	--	$\bar{1}.0$	—	Turner, H.J., et al. 1948
Barnacles Nauplii	MT	10	--	$\bar{0}.5$	--	Waugh, G.P., 1964
" "	No growth	10	--	$\bar{1}.0$	—	" " "
" "	LC-12	2,880	--	$\bar{0}$-0.25	--	Davis, M.H., and Coughlan, J., 1978
" "	LC-58	2,880	--	$\bar{0}.8$-1.0	—	" " "
" "	LC-62	2,880	--	7$\bar{1}.0$	--	" " "
Gammarus lig. (Amphipod)	LC-25	180	--	$\bar{2}.5$	—	Meldrim, J.W., et al. 1974
Melita Nitide (Amphipod)	LC-50	120	--	$\bar{2}.5$	—	McLean, R.I., 1973
" "	MT	5	—	$\bar{2}.5$	--	" " "
Corophium Sp. (Amphipod)	NE	410	--	1$\bar{0}.0$	--	Gentile, J.H., 1972
mphipod Juvenile	LC-50	5,760	--	$\bar{0}.687$	--	Thacher, T.O., 1978
Amphipod	LC-50	5,760	--	$\bar{0}.145$	--	Thacher, T.O., 1978
Blue crab	LC-50	1,140	--	1$\bar{0}.0$	--	
" "	LC-50	5,760	--	$\bar{0}.10$	--	Patrick, R., and McLean, R., 1970
Shape Crab	LC-50	5,760	--	$\bar{1}.418$	--	Thacher, T.O., 1978
Sand Shrimp (larvae)	LC-55	10	—	10.0	--	Gentile, J.H., et al. 1974
" "	LC-42	5	--	$\bar{5}.0$	--	" " "
Sand Shrimp	LC-50	700	--	$\bar{0}.15$	--	Patrick, and Mclean,R. 1970
Shrimp	LC-50	5,760	--	0.090	--	Thacher, T.O., 1978
Shrimp (Juvenile)	LC-50	5,760	--	$\bar{0}.134$	--	Thacher, T.O., 1978
Coonstripe Shrimp	LC-50	5,760	--	$\bar{0}.178$	—	Thacher, T.O., 1978
Grass Shrimp	LC-98	180	--	$\bar{2}.5$	--	McLean, R.I., 1973
Mysic Shrimp	LC-50	5,760	--	0.162	--	Thacher, T.O., 1978
Bugula Sp.	LC-100	2,880		2.5	--	Turner, H.J., et al. 1946
" "	LC-100	1,440	--	$\bar{1}0.0$	--	
Sea Urchin	DR-94-100%	5	--	0.125	--	Muchmore, P., and Epel, D., 1973
hiuroid	DR-22	5	--	$\bar{0}.40$	--	" " "

Species	Endpoint	(Min.) Exposure Time	Test Temp.	(Type) Conc. mg/L	Test Types	Reference
Echiuroid	DR-100%	5	--	$\bar{0}.40$	--	Muchmore, P., and Epel, D., 1973
Bottyllus Sp.	LC-100	1,440		10.0	--	Turner, H.J. , et al. 1948
Molgula Sp.	LC-100	4,320		1.0	--	
Fish						
Yellowtail Flounder	LC-50	1,440	--	$\bar{0}.1$	--	Gentile, J.H., et al. 1974
Plaice (Larvae)	LC-50	5,760	--	(-) 0.028	--	Alderson, R., 1970
"	LC-50	460	--	(-) 0.05	--	" " "
"	LC-50	75	--	(-) 0.075	--	" " "
"	MT	4,320	--	$\bar{0}.25$	--	" " "
Winter Flounder	LC-50	15	--	$\bar{2}.5$	--	Gentile, J.H., et al. 1974
" "	LC-50	0.3	--	$1\bar{0}.0$	--	" " "
Winter Flounder eggs	LC-0	20	--	$1\bar{0}.0$	--	" " "
Pink Salmon	LC-50	5,760	--	$\bar{0}.05$	--	Holland, E.A., et al. 1960
Pink Salmon	LC-50	7.5	13.6	$\bar{0}.5$	--	Stober, Q.J., and Hanson, C.H., 1974
Pink Salmon	LC-50	15	13.6	$\bar{0}.25$	--	
Pink Salmon	LC-50	5,760	--	(TRO) 0.023-.052	--	Thacher, T.O., 1978
Coho salmon	LC-50	7,200	--	0.08	--	Holland, E.A., et al. 1960
Coho salmon	LC-50	5,760	--	(TRO) 0.032	--	Thacher, T.O., 1978
Chinook Salmon	Distressed	60	--	0.10	--	Holland, E.A., et al. 1960
Chinook Salmon	MT	130	--	0.25	--	" " "
Chinook Salmon	MT	23	--	1.0	--	" " "
Chinook Salmon	LC-50	7.5	11.7	0.5	--	Stober, Q.J., and Hanson, C.H., 1974
Chinook Salmon	LC-50	30	11.7	0.25	--	
Young Salmon	MT	33,123	--	0.05	--	Holland, E.A., et al. 1960
Chinook Salmon	LC-50	5,760	--	0.038- 0.65	--	Thacher, T.O., 1978
Atlantic Silverside	LC-50	90	--	0.58	--	Engstrom, D.G., and Kirkwood, J.B., 1974
" "	LC-50	30	--	1.20	--	
" "	LC-50	5,760	--	0.037	--	Bellanca, M.A., and Baily, D.S., 1977
Atlantic Silverside (eggs)	LC-50	2,880	--	0.30	--	Morgan, R.P., and Prince, R.D., 1977

Species	Endpoint	(Min.) Exposure Time	Test Temp.	(Type) Conc. mg/L	Test Type	Reference
Tidewater Silverside	LC-50	2,880	--	0.21	--	Morgan, R.P., and Prince, R.D., 1977
Blueback herring	LC-50	60	--	0.67	--	Engstrom, D.G., and Kirkwood, J.B., 1974
Blueback herring	LC-50	15	--	1.2	--	
Blueback herring (eggs)	LC-50	4,800	--	0.33	--	Morgan, R.P., and Prince, R.D., 1977
Blueback herring (Larvae)	LC-50	2,880	--	0.24	--	" " "
Atlantic Menhaden	LC-50	60	--	$\bar{0}$.22	--	Fairbanks, R.B., et al 1971
" "	LC-50	10	--	$\bar{0}$.70	--	" " "
" "	LC-50	300	--	$\bar{0}$.21	--	Engstrom, D.G., and Kirkwood, J.B.,1974
" "	LC-50	30	--	$\bar{1}$.2	--	
(Larvae) Atlantic Menhaden	LC-0	3	--	$\bar{0}$.5	--	Hoss, D.E., et al. 1974
Threespine Stickleback	LC-50	5,760	--	$\bar{0}$.09-0.13	--	Esvelt, I.A., et, al. 1972
" "	LC-50	5,760	--	$\bar{0}$.167	--	Thacher, T.O., 1978
White Catfish	LC-50	2,880	--	$\bar{0}$.1	--	Esvelt, I.A., et al. 1972
Golden Shiner	LC-50	5,760	--	$\bar{0}$.03-0.23	--	" " "
Flounder	MT	5	--	$\bar{0}$.3	--	Hoss, D.E., et al. 1974
Striped Mullet (Juveniles)	MT	5	--	$\bar{0}$.3	--	" " "
Miscellaneous Marine Fish	Irritant Response	<1	--	$\bar{1}$.0	--	Hish, R.W., et al. 1953
White Perch	Death	480	--	(TRC) 0.8	--	Block, R.M., et al. 1978
White Perch (eggs)	LC-50	4,560	--	0.27	--	Morgan, R.P., and Prince, R.D., 1977
White Perch (larvae)	LC-50	1,440	--	0.31	--	" " "
Shiner Perch	LC-50	5,760	--	0.071	--	Thacher, T.O., 1978
English Sole	LC-50	5,760	--	0.073	--	Thacher, T.O., 1978
Pacific Sand Dance	LC-50	5,760	--	0.082	--	Thacher, T.O., 1978
Pacific Herring	LC-50	5,760	--	0.065	--	Thacher, T.O., 1978
Striped Sea Bass	LC-50	2,880	--	0.20	--	Morgan, R.P., and Prince, R.D., 1977

APPENDIX D

SUMMARY OF CHLORINE REACTION PRODUCT EFFECTS ON FRESHWATER AND MARINE ORGANISMS

Species	Endpoint	Exposure Time	Temp. °C	Test Type	Compound	mg/L Conc.	Reference
(Invertebrates)							
Daphnia magna	LC-50	48 hr.	--	S, U	1,2 - DCB	2.44	EPA 1978
" " "	LC-50	48 hr.	--	S, U	1,4 - DCB	11.0	EPA 1978
Midgel *Tanytarsus dissimilis*	LC-50	48 hr.	--	S, U	1,2 - DCB	11.760	EPA 1978
" " " "	LC-50	48 hr.	--	S, U	1,4 - DCB	13.0	EPA 1978
(Vertebrates)							
Rainbow trout	LC-50	96 hr.	--	FT, M	1,2 - DCB	1.58	EPA 1980
" " "	LC-50	96 hr.	--	FT, M	1,4 - DCB	1.12	EPA 1980
Fathead minnow	LC-50	96 hr.	--	FT, M	1,4 - DCB	4.0	EPA 1980
" " "	ELS	96 hr.	--	FT, M	1,2 - DCB	1.6-2.5	EPA 1980
" " "	ELS	96 hr.	--	FT, M	1,4 - DCB	0.56-1.04	EPA 1980
.uegill	LC-50	96 hr.	--	S, U	1,2 - DCB	27.0	Dawson 1977
" "	LC-50	96 hr.	--	S, U	1,2 - DCB	5.59	EPA 1978
" "	LC-50	96 hr.	--	S, U	1,4 - DCB	4.28	EPA 1978
(Plants)							
Alga, *Selenastrum capricornutum*	EC-50 chlorophyll *a*	96 hr.	--	--	1,2 - DCB	91.6	EPA 1978
" " " "	" " " "	96 hr.	--	--	1,4 - DCB	98.1	EPA 1978
" " " "	EC-50	96 hr.	--	--	1,2 - DCB	98.0	EPA 1978
	cell number	96 hr.	--	--	1,4 - DCB	96.7	EPA 1978

ELS = embryo larval stage
M = measured
S = static
U = unmeasured
FT = flow through

Species	End point	Exposure Time	Temp. °C	Test Type	Compound	mg/L Conc.	Reference
(Invertebrates) Mysid shrimp Mysidopsis bahla	LC-50	96 hr.	--	S, U	1,2 - DCB	1.97	EPA 1978
" " " "	LC-50	96 hr.	--	S, U	1,4 - DCB	1.94	EPA 1978
Polychaeteworm Polydorm webster	65% emergence from oysters	3 hr.	--	--	1,2 - DCB	100.00	Mackenzie, Shearer 1959
Nerlrs sp.	70%	3 hr.	--	--	1,2 - DCB	100.00	" " " " "
P. webster	55%	3 hr.	--	--	1,4 - DCB	100.00	" " " " "
P. Nerels	100%	3 hr.	--	--	1,4 - DCB	100.00	" " " " "
Tidewater Silverside Menidia beryllina	LC-50	96 hr.	--	S, U	1,2 - DCB	7.30	Dawson
Sheepshead minnow	LC-50	96 hr.	--	S, U	1,2 - DCB	7.66	EPA 1978
Cyprinodon variegatus	LC-50	96 hr.	--	S, U	1,4 - DCB	7.40	EPA 1978
Clam mercenaria mercenaria (embryo)	LC-50	48 hr.	--	--	1,2 - DCB	>100.	Davis, Hindu 1969
(larval)	LC-50	12 day	--	--	1,2 - DCB	>100	" " " "
Alga, Skeletanema costatum	EC-50 chlorophyll a	96 hr.	--	--	1,2 - DCB	44.2	EPA 1978
" " "	" "	" "	--	--	1,4 - DCB	54.8	EPA 1978
" " "	EC-50	96 hr.			1,2 - DCB	44.1	EPA 1978
	cell number		--	--	1,4 - DCB	59.1	EPA 1978

Species	End point	Exposure Time	Conc. mg/L	Chemical	Test Type	Reference
Plants						
Alga, Skeletonema lostatun	Chlorophyll a LC-50	96 hr.	3.270	4, chloro	--	EPA 1978
Skeletonema lostatun	EC-50 cell count	96 hr.	3.560	4, chloro	--	EPA 1978
Skeletonema lostatun	EC-50 cell count	96 hr.	0.890	2,4,5 tri	--	EPA 1978
Skeletonema lostatun	EC-50 cell count	96 hr.	0.960	2,4,5 tri	--	EPA 1978
Skeletonema lostatun	EC-50 Chloro a	96 hr.	0.440	2,3,5,6 tetra	--	EPA 1978
Skeletonema lostatun	EC-50 cell count	96 hr.	0.550			
Skeletonema costatum	25% cell inhibition	7 days	>8.0	4 chloro	S, U	Erickson
Skeletonema costatum	50% cell inhibition	7 days	>8.0	4 chloro	S, U	Erickson
Skeletonema costatum	25% cell inhibition	7 days	8.0	2,4,6 tri	S, U	Erickson
Skeletonema costatum	50% cell inhibition	7 days	8.0	2,4,6 tri	S, U	Erickson
Thalassiosiru pseudonunu	25% cell inhibition	7 days	>8.0	4 chloro	S, U	Erickson
Thalassiosiru pseudonunu	50% cell inhibition	7 days	>8.0	4 chloro	S, U	Erickson
Thalassiosiru pseudonunu	25% cell inhibition	7 days	2.0	2,4,6 tri	S, U	Erickson
Thalassiosiru pseudonunu	50% cell inhibition	7 days	4.0	2,4,6 tri	S, U	Erickson
Isochrysis galbanu	25% cell inhibition	7 days	>8.0	4 chloro	S, U	Erickson
Isochrysis galbanu	50% cell inhibition	7 days	>8.0	2,4,6 tri	S, U	Erickson
Isochrysis galbanu	25% cell inhibition	7 days	0.25	2,4,6 tri	S, U	Erickson
Isochrysis galbanu	50% cell inhibition	7 days	0.50	2,4,6 tri	S, U	Erickson

APPENDIX E

SUMMARY OF TOXIC EFFECTS OF OZONE ON AQUATIC ORGANISMS

Species	Residual Ozone (mg/L)	Summary of Effects	Reference
Bluntnose Minnow	0.03	Slight loss of equilibrium	Hubbs (1930)
	0.09	Death (56 min. exposure)	"
	0.25	Loss of equilibrium (25 min), then death at unknown time	"
	0.50	Loss of equilibrium (10 min.)	"
	0.50	Death, time unknown	"
	1.25	Near death (20 min.), experiment stopped	"
Fathead minnow	0.2-0.3	100% Death (1-3 hours)	Arthur, et al (1975)
Fathead minnow			
" Larvae	<0.1	<C-50 (0.5-5 min)	Coler & Asbury
" Eggs	1.10	<C-50 (160 min)	" 1980
Fathead Minnow	0.058	No mortality (11 days)	Ward et al 197
" "	0.47	No mortality (15 days)	"
" "	0.225	100% dead 20 hours	Ward et al 197
" "	0.059	No mortality 27 hours	"
" "	0-0.016	No mortality 330 days	Ward et al 197
larvae and eggs	0-0.013	No mortality 60 days	"
larvae, egg, fish	0.016-0.01	No growth or reproductive effects	"
adult fish	0.001-0.003	No mortality 330 days	Ward et al 197
Central Common Shiner	0.03	Slight loss of equilibrium, survived	Hubbs (1930)
"	0.25	Died, time unspecified	"
"	1.25	Near death (70 min), experiment terminated	"
"	1.84	Death (42 min)	"
Northern Common Shiner	0.003	40% mortality (4 days)	Ward et al 1977
" " "	0.016	No mortality 96 hr.	Ward et al 197
Pugnose Shiner	0.016	No mortality 96 hr.	Ward et al 197
Spottail shiner post larvae	1.22	24-h <C50	Colert Asbury
Rosyface Shiner	0.03	Irritated but survived	Hubbs, 1936
"	1.25	Near death (20 min), experiment terminated	" "
Western Blacknose Dance	0.03	Irritated, but survived	" "
	1.25	Loss of equilibrium, (11 min) Experiment terminated	" "
Northern Creek Chub	0.03	Irritated, survived	" "
"	1.25	Dead (4 hrs.) 100%	" "

Species	Residual Ozone (mg/L)	Summary of Effects	Reference
Rainbow Trout	0.01-0.06	100% Mortality (4 hrs.)	Rosenlund, 197
" eggs	< 0.10	Increased hatchability	Benolt & Matlin 1966
Juvenile	0.0093	96 hrs. <C-50	Wedemeyer et al 1979
" "	0.005	Damage to gill tissue	" "
" "	0.002	Little damage to gills	" "
Rainbow Trout	0.010	Normality 48 hrs.	Ward et al 197
" " larvae	0.19-0.31	<C-50 24 hrs.	Coler & Ashbur 197
Lake Trout	0.016	No mortality 96 hrs.	Ward et al 197
"	0.322	100% Mortality 5 hrs.	" " "
"	0.234	" " 42 min	Ward et al 197
"	0.048	" " 50% diluted 4.4 hr	" "
"	0.088	100% Mortality 21 hrs.	" "
"	0.126	" " 61 min	" "
Brown Trout	0.008	10% Mortality 48 hrs.	" "
	0.018	No mortality no time	Ward et al 197
Gold Fish	0.288	100% Mortality 62.7 hr.	Ward et al 197
" "	0.007	No mortality 96 hrs.	" " 197
" "	0.038	" " no time	" " "
" "	0.185	" " 7 days	" " "
Splake (Salvelinus pp)	0.007	5% Mortality 48 hrs.	" " 197
Chinook Salmon	0.008	5% Mortality 48 hrs.	" " 197
" "	0.018	No mortality no time	" " 197
Coho Salmon	0.010	" " 48 hrs.	" " 197
Bluegill	0.002	No mortality 96 hrs.	" " 197
" larvae	<0.10	<C-50 0.5-2 min	Coler & Ashbur 198
" "	0.33	<C-50 24 hrs.	" " (1980
Bluegill	0.06	<C-50 24 hrs.	Paller and Heidinger 1979
"	0.32	<C-50 30 min x 6, 8 hrs. apart	Paller and Heidinger 1979
Large Mouth Bass	0.012	No mortality 72 hr.	Ward et al 197
White Sucker egg	1.43	<C50 80 min	Coller & Ashbu 1980
Yellow Perch larvae	<0.10	<C50 2-5 min	" "
" " egg	1.57-1.98	<C50 - 80 min	" "
" " larvae	0.21	<C50 24 hrs.	" " (
" " egg	72.06	<C50 "	" " (
White Perch	0.22	<C50 96 hrs.	Rosenkranz (1978)
" "	0.1-0.15	low blood pH 96 hr. Hematocrit (Blood Changes)	
"	0.01-0.15	Cell damage in gills 24 hr. reversable	" "
American Shad eggs	0.39	<C50 24	(19 Coler & Ashbur

Species	Residual Ozone (mg/L)	Summary of Effects	Reference
Channel Catfish			
" larvae	0.47	24 hr. <C50	Coler & Ashbur
" eggs	4.0	<C50 24 hr.	(19)
Invertebrates			
Cray Fish	1.16	100% Dead 35 hrs.	Hubbs, 1930
Unspecified Planktonic and Bottom Invertebrate	1.25	100% Dead 35 hrs.	" "
Daphnia Mayna	0.030	30% Mortality 96 hrs.	Ward et al 197
Stone Flies	0.005	No mortality 2 days	Ward et al 197
Caddisflies	0.009	30% Mortality 2 days	" "
Amphipod *Gammarus sp*	0.009	No mortality 2 days	" "
Isopods *Ascellus sp.*	0.009	No mortality 2 days	" "
Snail *Goniobasis livescers*	0.005	No mortality 2 days	" "
Copepod	0.001	No mortality 2 days	" "
Cladoceran Simocephalus serrulatus	.001	<C,50 96 hrs.	" "
Marine & Esturiank			
Americanoyster eggs	<0.20	genetic changes Fertilization defects, cell abnormalities	(1973) Meleaw et al
Barnacles *Balanus sp*	0.4-1.0	Death several days - 7 days	Mangum, P.L & McIlhenny(19
Phytoplawktow *Skeletonema costatum* *Chlorella sp.* *Nannochloris lutheri*	0.10	Lethal 5 min	Toner and Brooks (1975)
Crab Zoea	0.08	(5 min exposure) 0-20% Mortality after 24 hrs 30-40% Mortality after 48 hrs	" "
Crab megalpos	0.2	100% Mortality after 24 hrs (1 min exposure)	" "
Atlantic Silverside	0.08-0.2	100% Dead (30 min)	" "
Striped Bass eggs	0.05-0.1	12 hr exposure - Delayed hatch	Richardson et al 1978